THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—19TH YEAR. SYDNEY, SATURDAY, NOVEMBER 5, 1932. No. 19.

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ANGINA PECTORIS AS AN ALLERGIC MANIFESTATION, AND OTHER OBSERVATIONS ON THE ALLERGIC STATE AND ITS TREATMENT.

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THIS paper is intended to suggest that certain symptoms, not commonly so regarded, may sometimes be allergic in origin, and to draw attention to some points in the diagnosis and treatment of the allergic state.

I include as "allergic" all "abnormal reactions" to foreign proteins, whether due to congenital idiosyncrasy alone or enhanced by antecedent sensitization, when they are distinguished as "anaphylactic". Barber and Oriel[1] use the term allergy for "all forms of hypersensitiveness" to protein, and that is, I think, its common use at the present time. The abnormal reactions are manifest in the skin as vascular dilatation with wheal formation, and in the viscera as spasm of involuntary muscle, the most familiar symptoms ascribed to them being urticaria and asthma.

Many other symptoms have been described from time to time as allergic. Freeman,[2] for instance, gives as an admittedly incomplete list, rhinorrhœa, asthma, urticaria, diarrhœa and vomiting, arthritis, colitis and nephritis, also he has drawn attention to the great importance of trauma in determining the organ in which the symptoms occur. There are, of course, many other causes of such symptoms, but allergy is coming to take a definite place in ætiology, and the diagnosis and treatment of the condition are clinically important.

Specific diagnosis, as by skin reactions, which must precede specific desensitization, is often impracticable; and it is important to have a means of non-specific diagnosis of allergy and also a general method of desensitization available against all proteins. Peptone has been in use for the latter purpose for some years; instances of its successful use are given below.

For the clinical diagnosis of allergy it is possible to argue as follows: Urticaria and asthma, very different symptoms, are both relieved by subcutaneous injection of adrenaline, and both are recognized as allergic manifestations, therefore in allergic patients any fresh symptom which is relieved by adrenaline is probably allergic likewise. I have thus come to regard the injection of adrenaline as a rough therapeutic test for allergy, and if relief is so obtained, I proceed to the further therapeutic test of desensitization with peptone. Relief by desensitization I regard as proof of the allergic nature of any symptom.

As regards the viscera, it seems at first sight probable that the allergic symptoms depend on an incoordination between the vagus and sympathetic nerves, overaction of the former, and depression of the latter. The asthmatic spasm, for instance, may be relieved either by paralysing the vagus nerve-endings with atropine, though in practice this is not very effective, or by stimulating the sympathetic, that is by injecting adrenaline, which is much more effective. This being the case, then other symptoms which could be explained by a similar incoordination seem also likely to be allergic in origin and to be suitable for treatment by adrenaline and desensitization, especially in known allergic subjects. This in practice makes a good rule of thumb for diagnosis and treatment, and it has been followed in the cases here reported.

Angina pectoris (Cases I and II) and biliary colic (Case VIII) both came into this category; thus Anrep and Segall[3] state that experiments demonstrate the presence of vaso-constrictor fibres to the coronary blood vessels in the vagi, and vaso-dilator fibres in the sympathetic nerve, in the heart-lung preparation in the dog. If allergy were to lead to overaction of the vagus in this situation in man, it would presumably diminish the blood supply to the heart and predispose to anginal pain, which should be relieved by the injection of adrenaline. This, in the only cases in allergic subjects so far tried, has proved to happen in practice.

Again, the gall-bladder is supplied by the vagus and sympathetic nerves. Experimentally, if the vagus is slightly stimulated, the fundus contracts and the sphincter of Oddi relaxes, as in normal emptying, but under strong stimulation both fundus and sphincter contract (Bain[4]). If this were to occur in man from an allergic cause, the conditions required for visceral pain would be present and presumably biliary colic would result, and the use of adrenaline to relieve it would be rational. Cases have occurred which appeared to have this origin.

It is, of course, recognized that *angina pectoris* and biliary colic are generally due to other causes than allergy, but if the vago-sympathetic hypothesis were sound, they might reasonably be expected to occur in allergic subjects, either spontaneously or after trauma, and should then respond to adrenaline.

Briefly, the suggestions for the diagnosis of allergy are as follows: Unexplained symptoms in allergic subjects are likely to be of an allergic nature; symptoms which could be explained by an overaction of the vagus and depression of the sympathetic are particularly likely to be allergic, and such symptoms are frequently relieved by injection of adrenaline and reduced in frequency by desensitization.

The vago-sympathetic hypothesis is critically examined at the conclusion of the paper.

Angina Pectoris.

CASE I.—W.H., aged forty, a male patient, was admitted to Dunedin Hospital on August 16, 1929, with influenzal bronchopneumonia. Physical signs were present on the right side, with considerable pleurisy. The temperature become normal in seven days, and the pulse rate was 80 per minute in ten days. He was discharged four weeks after admission, apparently quite recovered, but he did not feel well and attended the out-patient department, and complained of pain, for which he was readmitted on October 4, 1929. The following notes were made at the time:

> Lately he has experienced retrosternal and precordial pain, and also a feeling of impending dissolution. The pain behind the sternum shoots up into the neck and he feels as though he were going to choke, he has to sit up to get his breath. The pain is always worse at night; it is sharp and stabbing, runs round the left side of the chest, and radiates down the left arm as far as the wrist; over the ventral aspect of the left wrist the pain is again sharp and stabbing, and it is also felt in the left axilla and shoulder. He is obviously worried about himself, and wonders whether he is going to die, although he feels physically strong and fit.

Examination of cardio-vascular system on readmission to hospital showed that the heart was not enlarged, no murmurs were present. The pulse rate was 100 (it fell to 80 in a few days), the pulse was regular. The electrocardiogram was normal but for accentuated *T* wave in Lead II. The systolic blood pressure was 144, and the diastolic pressure 70 millimetres of mercury. The specific gravity of the urine was 1·020; it contained no albumin.

A typical bout of *angina pectoris* is thus described without any cardiac condition to account for it. The patient, however, stated that for six or seven months he had had severe headaches at intervals.

The pain is felt mainly behind the eyes, and shoots through to the occiput, it is practically confined to the left side of the head. Headaches are always preceded by a warning which occurs about half an hour before their onset, and takes the form of some disturbance of vision, such as photopsia, chromatopsia, *muscæ volitantes*, scintillating scotomata or fortification spectra. Vision is blurred, and he complains of "fern leaves" in front of his eyes. Headaches are so severe at times that they are accompanied by vomiting.

This is an equally typical account of attacks of migraine.

Two minims of adrenaline were given for the angina with slight relief; there was complete relief with 0·015 gramme (a quarter of a grain) of morphine, with 0·6 milligramme (one one-hundredth of a grain) of atropine; one further attack of angina was aborted by 0·3 mil (five minims) of adrenaline.

On July 31, 1930, the patient was reexamined. Occasional attacks of anginal pain occurred, generally when the patient was at rest, never when he was at work; sometimes he complained of a "smothering" sensation. He claimed recently to have worked thirty-two hours continuously keeping fires going in a ship in port; no disability resulted. The heart rate was about 140 under examination, and it had rather a tic-tac rhythm; he was nervous at having been sent for to be examined.

This patient is an unlikely subject for "true" angina due to myocardial defect, and the history of migraine suggests a possibly allergic origin, which

is confirmed by the response to adrenaline. He did not attend regularly for desensitization.

The possibility of anginal pain being due to allergy may have some bearing on the cause of cardiac pain, a matter not yet completely settled. Cowan[5] in his St. Cyres Lecture on fibrosis of the heart, set out at some length the views which are best accepted at the present time. He attributes the pain to anoxæmia of the heart-muscle, induced by coronary obstruction from any cause, as was suggested by Allan Burns and also by MacWilliam, on the analogy of the pain induced in a limb by exercise after it has been rendered ischæmic by an elastic bandage. No doubt a similar embarrassment occurs in both cases, although it is not quite satisfactory to compare pain in a limb with pain in the heart, which, according to Waugh[6] can be grasped under a local anæsthetic without pain, and without its rate being altered. Parkinson and Bedford[7] in a recent paper refer to the morbid anatomy of the heart in anginal cases as "ischæmic fibrosis of the myocardium due to obliterative arteriosclerotic changes in the coronary field", and they insist that the coronary arteries are the source of anginal pain.

If the coronary vessels of the human heart are supplied by the vagus and sympathetic, as Anrep and Segall find to be the case in the dog, then an allergic overaction of the vagus would be likely to induce anginal pain, which should be capable of relief with adrenaline. These observations appear to be in line with the prevailing views as to the coronary origin of anginal pain.

CASE II.—Mrs. H., aged sixty-two years, had good health till two years ago, when she consulted her doctor, who she said diagnosed "blood pressure", which was 160 millimetres of mercury (systolic) on admission. On exercise she suffers from pain in the precordium "like a gripping" which moves up to the left shoulder and down the left arm "like a rheumatism". During the same period she has had unilateral headache, on either side of the head, when she sees lights "light bright stars"; the headache is relieved by vomiting. An attack of migraine immediately before her admission to hospital had ended in a left hemiplegia, for which she was sent in. She showed all the reflex changes usual in hemiplegia, but the attack was quite transient, and in two days no trace of it was to be found. Her heart had a tic-tac rhythm and a loud aortic second sound.

I take it that this patient's myocardial degeneration induced an allergic *angina pectoris* on exercise, if there were no strain on her heart migraine was an alternative manifestation. I have met with two other comparable cases.

One occurred in a woman of about forty, who has had cardiac symptoms for more than ten years. She is subject to gross paroxysmal disturbances of rate and rhythm of the pulse, sometimes going on to syncope, and there are abnormalities in the electrocardiogram; she certainly has myocardial disease. She has been subject for many years to migraine, and sometimes to nocturnal asthma; the latter is relieved by adrenaline. Recently she has described attacks of pain which are typically anginal, and which, she claims, are also relieved by adrenaline.

In the light of the cases described I take it that this patient is an allergic subject who also suffers from heart disease, and that the anginal manifestation of allergy is determined by the trauma of her myocardial lesion. Desensitization has not been tried. Quite recently she has had abdominal pain, suggesting biliary colic to another doctor, but Graham's test revealed no abnormality.

The other case is that of a man aged fifty-two, a chronic epileptic, who describes a pain which one would take for *angina pectoris*, but who shows no other indication of heart disease. A reference is made below to cardiac disturbance as an epileptic variant.

These tentative diagnoses were made on the assumption that in a person with recognized allergic symptoms, a new symptom which is explicable as a depression of the sympathetic is likely to have the same cause. The following are a few instances of patients who show multiple allergic phenomena.

Multiple Allergic Symptoms.

CASE III is one of asthma, urticaria and albuminuria. M.S., a female patient, aged thirty-six years, gave a history of pain and stiffness of several joints which had subsided; she was not seen in this condition. She had severe asthma and also albuminuria; the albumin was precipitated from her urine, filtered and dried, and a distinct wheal was obtained with the powder in a scarification test. [This (1923) was long before proteose reactions had been demonstrated in the urine of allergic patients;[8] the suggestion at the time was that the offending protein was in process of elimination by this means.] This patient showed urticaria very readily, and on the injection of 0·25 cubic centimetre of 5% peptone was covered with wheals from head to foot.

CASE IV is one of asthma, migraine and epilepsy. C.B., a middle-aged male patient, was first seen in Dunedin Hospital with asthma, and some years later was readmitted with a diagnosis of epilepsy; he suffered from attacks closely resembling *petit mal*, and complained also of hemicrania. He gave a history of an injury to the head through a severe fall on tramlines in childhood.

In this case also the trauma may have determined the cerebral symptoms. Many textbooks insist on the association of migraine and epilepsy.

CASE V is one of migraine and colonic spasm. C.F., a female patient, aged sixty-two years, was an intelligent ex-school mistress, who had long suffered from "bilious attacks", for which she had been treated with stomachics. The attacks were associated with hemicrania and responded well to desensitization. She complained later of attacks of painless diarrhœa, coming and going without any recognizable cause; only one opportunity occurred for the use of adrenaline, and on this occasion the attack was delayed for several hours beyond its usual period of onset. (Her migrainous attacks are discussed as Case XIII *infra*.)

CASE VI is one of eczema, migraine and hay fever. D.H., a male patient, aged forty years, had an unsuccessful injection for varicose veins one year previously; the leg became very much inflamed, with extensive eczema. Since then transient attacks of eczema have occurred in different parts of the body which come and go spontaneously; angioneurotic œdema of one orbit has occurred. This patient is subject to headache with attacks of vomiting, and suffered from hay fever in youth, another instance of allergic manifestations being determined in site by trauma. He was seen again a year later: he stated that he had peptone injections for three months, the lesions disappeared after three or four injections and there had been no recurrence except for a small patch on the forehead where the hat rubbed.

In all these cases there were histories of former symptoms which were of recognized allergic character, and fresh symptoms which were in most cases of similar nature; the diagnosis of "allergy" was the first to suggest itself. In the following two cases it was more speculative.

Joint Conditions.

CASE VII is one of "arthrosis". The patient was under the care of Dr. R. S. J. Fitzgerald, Oamaru, New Zealand. A.D., a female, aged forty-six years, had been ill for two months with a condition first diagnosed as rheumatoid arthritis. Pain and swelling occurred in the knees, wrists and joints of the fingers, but did not have the typical characters of that disorder. There was a history of an attack a year before for which she went to a spa, but the condition subsided spontaneously immediately upon arrival there without any treatment, and the patient was soon playing tennis without any indication of antecedent arthritis; this does not occur in any ordinary inflammatory lesion of the joints. With this history the diagnosis of allergy was made, desensitization was employed, and improvement immediately followed, though without the dramatic suddenness of the first recovery, and power was regained without any sign of lesion in the joints. She has remained free for two years.

Biliary Colic.

CASE VIII is one of urticaria, angioneurotic œdema and biliary colic. J.R., a middle aged man, first suffered from skin lesions during the War, which he attributed to handling camels. He returned to New Zealand and was admitted in 1923 to Dunedin Hospital for urticaria. Extensive skin tests were carried out, especially with various woods, as he is a carpenter by trade and "birch-itch", a form of sensitivity, is not uncommon among bushmen. No specific sensibility was detected. He was readmitted to hospital in 1930 with, apparently, typical biliary colic, but Graham's test failed to reveal an abnormality. The attack subsided spontaneously and completely, but angioneurotic œdema of one orbit and an attack of urticaria both followed. He then stated that these lesions are generally associated with his "stomach attacks". He derived some benefit from peptone injections.

I have found only one reference to biliary colic as an allergic manifestation. Graham, Cole, Copher and Moore[9] in their book on the gall-bladder state that intestinal allergy is probably rather frequently confused with cholecystitis, but this has received "almost no mention". I have recently reviewed a series of 100 cases of cholecystitis in Dunedin Hospital. In eleven of these colic had occurred, but no stone was present in the gall-bladder or ducts at operation; in two of the cases there was no evidence of cholecystitis. It appears then that colic without stone is not very rare and that it may occur without inflammation. The nerve supply of the gall-bladder has been briefly reviewed above. If, as is suggested, allergy were to induce overaction of the vagus and paresis of the sympathetic, and its incidence happended to fall on the gall-bladder, a condition of colic would follow. I am having all my patients with biliary colic injected with adrenaline, in hopes of giving relief.

Cases I, VII, and VIII are of a kind in which allergy is not likely to be the first diagnosis suggested, but the patients are all well worth submitting to the therapeutic test.

Epilepsy.

Several cases of migraine have been mentioned, one of which was associated with *petit mal*. The association of epilepsy and migraine is indeed familiar, and most students of allergy include epilepsy as one of its manifestations. Some points of interest in regard to both of these have arisen. Kinnier Wilson[10] in discussing "epileptic variants" postulates "periventricular fits", in which epileptic disturbances occur in many viscera, among them the heart. He describes palpitation and other subjective cardiac symptoms, but states that pain is rare, though he has met with a typical case, with radiation of pain down the left arm. I take this case to be parallel to Case I, described above. Kinnier Wilson mentions also the "sense of impending dissolution", which distressed my patient.

Mackenzie Wallis and Nicol[11] investigated a special type of epileptics recognized by Sir Maurice Craig. They are healthy persons inclined to obesity, not excitable; they do not react to bromide. They give positive reactions to various proteins and may be successfully treated by diet or oral administration of peptone. I do not think the cases quoted below belong to this type.

I have used peptone subcutaneously in many cases of epilepsy, following a suggestion by A. G. Auld.[12] Shortly after his letter was published a young girl who had recently begun to menstruate was admitted to Dunedin Hospital with fits which occurred only at the periods. Unfortunately the records of this case are imperfect, and the patient cannot be traced, but the facts are approximately as follows:

CASE IX. M.S., a female patient, aged fourteen years, had a fit for the first recorded time at her first menstrual period, the fits were repeated at her second period, and she was admitted to hospital and fits were observed at her third period. She was treated with peptone and no fit occurred at the fourth period, when she was discharged to the out-patient department and attended there for some months without a recurrence, but the exact time is not known.

It is of course no very uncommon thing for a female to have fits when menstruation begins, which are not repeated, so that this case was only an encouragement to investigate others.

I prescribed peptone for every available patient with epilepsy for some time, but, under the conditions of consulting practice, when the majority of patients are seen only once, reliable records of results are rarely obtained. I have used peptone upon several chronic epileptics of degenerate type, but they never derived the smallest benefit therefrom. Other, more favourable cases, but urgently demanding treatment, were spoilt for the purpose of therapeutic experiment in that the patients were given one or other of the recognized drugs, peptone being used only as an adjuvant. The following notes of a patient under the care of Dr. D. G. Radcliffe, Balclutha, New Zealand, have kindly been given me by him.

CASE X.—W.T., a male patient, aged thirty-four years, had his first known fit in March, 1928, and others in June and August, with *petit mal* attacks in addition. There was no known cause, and signs of cerebral tumour were carefully sought for. He was put on Gélineau's *dragées* and a salt-free diet, but the fits continued at intervals varying between one and four weeks. By May, 1930, he was thin, losing weight, shaky, slovenly in habits and dress, tremulous and careless. Peptone injections were begun at this time and were given at intervals of a week for four months. For three months he was free of fits, he has since had a few mild fits. He has put on more than a

stone in weight, and from being subnormal in intelligence and behaviour, he has become quite normal, neat, tidy and careful in habits, dress and appearance.

Unfortunately the therapeutic experiment is vitiated by the fact that he has taken a grain of "Luminal" a day; this drug, however, rarely produces any such marked effect.

Dr. Radcliffe, who uses peptone extensively in asthma, informs me that he has frequently found that patients who are improving under it, put on weight. Such a result as this is not going to be obtained in every case of epilepsy. It was the best result in my experience, but I have seen several which are comparable to it. If such a result can be obtained, it is surely well worth making the therapeutic experiment, and a patient manifesting other allergic symptoms would be the most likely to derive benefit.

CASE XI was one of epilepsy. D.C., aged fourteen years, a female patient, was under the care of Dr. R. S. J. Fitzgerald. She had had nocturnal fits for four years, increasing in frequency. The tongue was bitten and the urine was sometimes passed during a fit. Three to six attacks occurred every night and someone had to sleep with her. I advised peptone injections, and four months later Dr. Fitzgerald informed me: "She has had regular injections, at first weekly, then every two weeks and lately every three weeks. For the last three months she has had two or three mild attacks each week, but no severe spasms as before. She has gained weight and is much brighter mentally." She was also given "Luminal" 0·06 gramme (one grain) every night, but this had been given before without effect. The girl has a rather narrow palate and "Simian" hand, and I had not anticipated much benefit from the treatment.

Migraine.

Migraine is the condition in which I have had, personally the greatest number of successes in treatment with peptone. That migraine is "interchangeable" with asthma, epilepsy, urticaria and other allergic phenomena is agreed upon by most people interested in the subject. The typical attack is essentially "periodic", and consequently any treatment is difficult of accurate evaluation; its chief manifestations are unilateral headache, with scintillating scotomata or other subjective visual phenomena, and vomiting, after which relief is generally obtained. The variations and extensions of the symptoms need not be mentioned here. The mechanism of the attack remains obscure; it was very fully discussed at a meeting of the Sections of Neurology and Ophthalmology at the British Medical Association annual meeting in 1926. Dr. Edwin Bramwell[13] favoured intracranial vascular disturbance, probably in the meningeal vessels, since the brain substance is insensitive. He suggested that the pain might be due to local dilatation of the vessels, following arterial spasm. While he admitted that there was no proof that the cerebral arteries were regulated by a vasomotor mechanism, he drew attention to the obvious vascular changes in vessels outside the skull. Mr. W. H. McMullen[14] said that both spasm and dilatation of the retinal arteries were occasionally observed, and Dr. Gordon Holmes[15] was emphatic that no effect could be artificially produced upon the cerebral vessels by way of the sympathetic. He postulated a local swelling of the brain from imbibition of fluid, a condition, one would suppose, closely resembling urticaria. I have recently used adrenaline during migrainous headache with uniformly good results. I have given it in the manner prescribed by Hurst[16] for asthma; that is, I leave the needle in place and inject 0·06 mil (one minim) of a one in one thousand solution at a time, until relief is obtained, and am prepared to withdraw the needle at once if the headache should be aggravated. In any other part of the body outside the skull, the relief of the condition by this means would be easily explained. Everyone has seen urticarial swellings reduced by adrenaline; and if its general effect is to induce contraction of the arterioles, then it should relieve the local dilatation which Dr. Bramwell postulated as the cause of the pain following a previous spasm. Whatever the physiological action of the drug, I have found its use to be always satisfactory and often to give complete relief.

The following are a few cases of chronic migraine in which peptone has been successfully used.

CASE XII. L.G., a schoolmistress, aged twenty-nine years, was asthmatic in early life. She enjoyed good health till a year before treatment began, she then had prolonged nervous strain from her father's illness and death. Since then she had had "attacks" regularly at the end of the school week, generally on Saturdays; hemicrania, vomit, exhaustion and no visual change. After peptone treatment was begun there was immediate reduction in the severity of the attacks, but they were still frequent. After two months, she went free for two weeks at a time, and after three months she frequently had a month's freedom, and there was no vomiting. The improvement was not uniform; occasionally bouts used to occur. The injections were for a long time given at monthly intervals, and I pressed this patient not to attend unless she was satisfied that her condition was improved. She continued to attend for four years, insisting that if she refrained from treatment, the attacks recurred in a few weeks. During the last year, though I have attended her for other complaints, she has had no peptone and no headache of consequence. Here general health has much improved, and she has been distressed by her increase in weight.

CASE XII. C.F., aged sixty-two years, an ex-schoolmistress, was subject to attacks since the menopause; she suffered from hemicrania (either side), and complained of fortification spectra and of vomiting. She began treatment in October, 1929, and continued for six months, during which time she was free of attacks. Treatment was then suspended, but attacks recurred and further injections were given until about twelve months ago with the same relief. Since then there have been no further headaches.

CASE XIV. W.M., aged fifty years, a carpenter, complained of attacks of headache, vomiting and scintillating scotomata which began about seven years ago. These symptoms increased in frequency and severity, till the man was losing a day's work every week. I was called to see him on account of a supposed gastric condition, which was evidently migrainous. The attack which was then beginning to subside, was immediately terminated by adrenaline. He has been treated with peptone for twelve months, the attacks have been reduced to one a month, and have only on one or two occasions been severe enough to make him stop work.

I have found the majority of cases, a dozen or so, follow a course comparable to Case XIV, but

naturally such results are not invariable. This is shown by Cases XV and XVI.

CASE XV. J.R., a female patient, aged fifty-three years, complained of headache in bouts lasting twenty-four to thirty-six hours. She had suffered from these attacks all her life. They were generally unilateral, but might occur on either side; they were accompanied by nausea and occasional vomiting. Her condition improved under peptone treatment for about two months. After this the attacks recurred and increased in severity during a further three months of treatment; it was then abandoned.

CASE XVI. C.D., a male patient, aged thirty-nine years, complained of unilateral headache and vomiting. He had been subject to these attacks all his life, but they had been increasing in severity for five years. Severe attacks occurred every ten days, slighter ones occurred between the severe attacks. He underwent peptone treatment for about three months, but without any benefit.

Discussion.

The assumption of vago-sympathetic incoordination and of its correction by the injection of adrenaline is valuable in the clinical diagnosis of allergy; it remains to be considered whether it has any real physiological basis. As an hypothesis, it seems to be quite untenable in view of the work of Sir Thomas Lewis and his school on vascular reactions of the skin and of the cognate work more recently presented by Dr. H. H. Dale in his Croonian Lectures on chemical factors in control of the circulation.[17]

To summarize what is now widely recognized: Wheal formation occurs when histamine is pricked into the skin; it is due to a direct action of this substance on the blood vessels and the wheal is accompanied by a wide spread flush caused by an axone reflex.[18] The anaphylactic skin reaction depends on the release of histamine in the skin.[19] Whatever the action of adrenaline in reducing the wheal may be, it is clearly not the correction of any nervous incoordination.

In the viscera, the proved action of histamine demands the reconsideration of the whole of the allergic or anaphylactic phenomena, and even here the vago-sympathetic hypothesis could be maintained only in a very modified form. As long ago as 1921 Gideon Wells[20] stated that "it has now been established that isolated (sensitized) non-striated muscle tissue . . . will give strong contractions with . . . the specific antigen". Clearly no nerve influence whatever is here involved, but histamine is released; the chemical factor is paramount. Chemical factors have received increasing attention in physiology ever since the recognition of the nature and importance of hormones. Histamine and acetyl choline have received particular study. Injury to the skin releases histamine, the axone reflex releases a vasodilator substance, possibly acetyl choline, and Dale states that the specific changes in the heart rhythm produced by the stimulation of the vagus and sympathetic nerves respectively are due to the liberation of chemical principles.

Histamine is also present in the lung, and is released by injury (Harris[21]). It induces intense stimulation of plain muscle, particularly of the uterus and the bronchi; released by stimulus or injury of plain muscle, it will play a part in exciting or maintaining contractile reaction (Dale). One thinks immediately of allergic asthma in this connexion, and it is to be noted that in certain cases—in the liver of the dog—the action of adrenaline neutralizes that of histamine. Presumably, therefore, in allergy it is not an overaction of the vagus which is in progress, but the action of a chemical substance released by injury; vagus overaction is nevertheless very closely simulated in allergic states, and its apparent occurrence rightly suggests that diagnosis.

Dale's lectures indicate the extreme complexity of the chemical reactions involved and the very great difficulty in their interpretation; and it is manifest that no physiologist would accept anything so facile as the neutralization by adrenaline of either histamine or acetyl choline as an explanation of the clinical value of the former in the treatment of allergic symptoms. A clinician can only infer that the action of adrenaline is chemical and not nervous, but that does not solve the problems of diagnosis and treatment.

Be that as it may, no clinical observer will admit that in asthma, the visceral symptom which is most frequently ascribed to allergy, nervous, that is reflex, conditions can be ignored. Everyone knows how errors in diet will induce asthma in the susceptible, and I have myself known the symptom abolished by the extraction of crowned teeth, by the expulsion of a tapeworm and by the treatment of *pruritus ani*. An abnormally sensitive respiratory centre, accessible to impulses from many nervous surfaces, is often postulated in asthmatic subjects. Langdon Brown[22] fines this down to the "broncho-motor and vaso-motor portions of the vagus nucleus" and presents a diagram of the same and its connexions.

It is inherent in the conception of symptoms as disturbances of function, either by way of excess or defect, that a variety of causes may produce identical results, and in any discussion on asthma rhinologists, immunizators and psychiatrists will all claim cures. Anyone suffering from bronchitis who has a sensitive respiratory centre may get a reflex asthma, but if he is an allergic subject he is far more likely to exhibit that symptom, in fact the trauma in his lung is nearly certain to determine the allergic manifestation to that site. It is therefore just as important in the treatment of allergy by immunization to remove sources of reflex irritation as it is in the parallel case of vaccine therapy. If biliary colic really occurs as an allergic symptom, it will not be cured by desensitization if at the same time there are stones in the gall-bladder, and it is often clinically impracticable to determine how far such symptoms are allergic and how far reflex; the therapeutic tests are occasionally of use.

Relief by the injection of adrenaline only suggests the allergic state. No doubt the asthmatic spasm, whatever its origin, may be reduced by the injection of adrenaline, and this is no proof of

allergy. One might perhaps say that all allergic symptoms are relieved by adrenaline, but all symptoms relieved by adrenaline are not allergic. In the absence of reflex causes for such symptoms, however, desensitization is worth a therapeutic trial.

With regard to desensitization by peptone, Dale long ago suggested that the anaphylactic phenomena were immunity reactions between antigen and antibody occurring within cells; and he maintains that the release of histamine, now known to occur during the reaction, does not disprove his view. The reaction does not occur if the affected cells are desensitized, and when the specific protein cannot be identified, peptone may be used as a universal desensitizer, as suggested originally by Auld.[23] This worker was led to attempt the treatment of asthma by peptone injections by reasoning which I think was briefly as follows. The anaphylactic reaction in the guinea-pig closely resembles the asthmatic spasm in man, therefore asthma may be in some cases an anaphylactic reaction, though the offending protein may be difficult or imposible to find. An animal sensitized to a series of proteins is desensitized to all if it is desensitized to one, therefore there is such a thing as non-specific desensitization. Peptone is said to induce asthma in the guinea-pig, therefore peptone might be a suitable universal desensitizer in such cases. Auld described asthmatic patients successfully treated thereby. Later he suggested that "explosive-attacks" in other parts, in stomach, liver, central nervous system and cardio-vascular system might have a like origin and be susceptible to the same treatment. He quoted a case of migraine in which successful treatment by peptone was carried out; later again he mentioned epilepsy in the same connexion. He put forward the views that peptone produces in the body a poison which is identical with the anaphylactic poison, and that therapeutic doses of peptone induce tolerance to the anaphylactic poison, hence their value in treatment. This makes a sufficiently good working hypothesis for a clinician unversed in advanced biochemistry.

While I owe the suggestion of the use of peptone in allergic conditions to Auld, I have not followed his dosage nor his modifications by the addition of serum or agar. Brought up in Sir Almroth Wright's school of vaccine therapy, I incline to minimal doses in all attempts to induce active immunity, and I use peptone "to induce tolerance to the anaphylactic poison" precisely as if it were a vaccine. I use 5% peptone as prepared by the Bacteriological Department here, which is approximately half Witte's and half Armour's, and begin with 0·25 cubic centimetre of this solution. This is injected subcutaneously at intervals of a week and gradually increased until the dose which appears to be effective is found, and this is repeated as long as it gives good results. Sometimes 0·5 cubic centimetre is enough, I rarely exceed two cubic centimetres. The interval is gradually increased to two, three or four weeks. I find that in most of the successful cases the patients remain satisfied with a dose about once a month.

I prefer the subcutaneous route for two reasons. No very rapid result is to be expected from the method, and if two or three days are required for absorption, that is of no consequence and is perhaps desirable. Secondly, most of the patients visit me for treatment and I think it undesirable to give intravenous injections to ambulant patients; some persons are highly susceptible to peptone by this route. I have never seen the slightest local reaction or general intoxication from the doses which I employ.

Summary.

The varieties and clinical diagnosis of allergy with its suggestion of vago-sympathetic incoordination are discussed. Cases of *angina pectoris*, of multiple allergic symptoms, of epilepsy and migraine are described.

The vago-sympathetic hypothesis is further discussed and rejected, while its clinical value is appreciated.

The importance of reflex factors in allergic symptoms is emphasized and the rationale of desensitization by peptone is discussed.

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RELATIONS BETWEEN THE WEIGHT OF BREAST-FED INFANTS, THEIR ORDER OF BIRTH, AND THE YIELD AND COMPOSITION OF THEIR MOTHERS' MILK.

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COMPARATIVELY little attention seems to have been given to the study of possible correlations between the physical characteristics of human individuals and their order of birth. The attention which has been directed to the subject has been largely restricted to an examination of possible differences between the first and later children of a mother. The first born male of a family occupies a unique position in the social organization of many peoples. He is usually accorded a status carrying with it privileges not shared by his younger brothers. It is somewhat surprising that references to differences between the characteristics, especially physical, of first and later children are so infrequent in the literature dealing with the growth of children.

In the biblical writings there are, indeed, many instances in which the expression "first born" is used to connote the possession of superior or outstanding qualities of some kind. But no clue is given to the nature of the qualities which the first born, in the literal sense, might be supposed to show. Any distinguishing qualities of the first born male which may exist have been obscured by the fact that only approximately half the total number of first born males are first children. The occurrence of first born female children is usually not taken into account.

The principal observations made on this subject during the latter half of the last century have been collected by Vierordt (1906). These observations seem to be limited to measurements of the weight and length at birth of first as compared with the later children of a family. They agree in showing that the average weight and height at birth of first children are materially smaller than the values for later children. A relation between the birth weights of infants and the ages of their mothers has also been shown by one or two of these earlier workers. A relation of this kind may be expected, as the age of women increases with the number of children which they have borne. Some of this earlier work also shows that the birth weights of the last children of numerous families, or of older women, tends to decrease.

Among the more recent investigators Pearson (1914) has made similar observations. Murray (1924), Adair and Thielander (1925), and Harris (1925) have also found slight positive correlations between the birth weights of infants and the ages of their mothers or the order of their birth. By far the most comprehensive investigation of the subject is that by Mackinlay (1926) on the material, collected by numerous collaborators, which forms the subject of a lengthy monograph by Paton and Findlay (1926). These investigators agree with previous workers in finding a positive correlation between the weights and lengths of infants at birth and the ages of their mothers or the orders of their birth, and also a falling off in the size of the later children of large families or older mothers. This investigation included an examination of the heights and weights of infants up to their first year of age. It was found that by the time this age was reached the first children had more than made up for their initial deficit of size, and were both heavier and longer than later members of the families at the same age.

Another aspect of the relation between the physical characters of individuals and their order of birth was examined by Beeton and Pearson (1901). They found that the first children of families were, on the whole, definitely longer lived than their younger brothers and sisters.

In the present investigation attention was first directed to the existence of a relation between order of birth and weight at various ages as a result of an indication previously observed (Wardlaw and Dart, 1926) of a possible relation between the number of children a woman had borne and her yield of milk. The material discussed in the paper cited, and some additional material not published, have been examined to see whether any relation could be established between the food supply of breast-fed infants, their weight, and their order of birth. In particular it was desired to ascertain if the greater weight of the later breast-fed children of families of moderate size could be attributed to a more rapid growth associated with a more plentiful supply of milk.

The observations which have been made on the relation of the weight of infants at birth, or at various ages up to six months after birth, are in general agreement with those of the investigators cited. No evidence has been obtained to support the supposition that the relatively heavier infants of various ages are heavier because they have grown more rapidly as a result of the more plentiful supply of milk at their disposal. The same groups, with regard to order of birth, which were heavier at birth, were also heavier at the later stages of growth within the period mentioned.

Relative Weights of Infants at Birth.

The absolute and relative weights at birth of 275 normal infants born in Sydney are given in Table I. The absolute weights are given in metric units. They were recorded in avoirdupois units in the routine weighing of the infants at birth in a large maternity hospital. The series contains internal evidence that some of the weights may have an error as much as $\pm$ 60 grammes ($\pm$ two ounces). The effect of this error, however, will be to obscure any orderly relation which may exist between weight and order of birth, so that any such relation which appears, will do so in spite of, not because of the occurrence of such errors.

TABLE I.

Absolute and Relative Birthweights of Sydney Infants of Different Orders of Birth and Ages of Mothers.

MALES.

Number.	Birth Order.	Weight.		Mother's Age.
		Kilograms.	Relative.	
1	1	3·06	86	29
3		3·40	95	19
5		2·49	70	21
7		4·54	126	19
9		2·95	82	22
11		2·27	63	23
13		4·08	113	22
15		3·63	101	24
17		4·08	113	22
19		3·15	88	25
21		2·95	82	21
23		3·29	91	35
25		2·72	76	19
27		3·97	110	18
29		3·63	101	27
31		3·40	95	18
33		3·63	101	21
35		3·46	96	24
37		3·52	98	15
39		3·63	101	19
41		3·63	101	18
43		3·40	94	21
45		3·63	101	22
47		3·18	88	18
49		3·40	94	23
51		2·41	70	36
53	2	3·97	110	29
55		3·63	101	22
57		4·11	114	31
59		3·46	96	21
61		5·22	145	24
63		2·95	82	31
65		2·95	82	25
67		3·63	101	19
69		2·72	76	23
71		3·52	97	17
73		3·69	107	25
75		3·97	110	17
77		3·27	92	18
79		3·40	95	27
81		3·18	88	20
83		3·40	95	22
85		4·65	129	23
87		4·54	126	25
89		3·35	93	22
91		3·80	106	28
93		4·36	121	20
95		3·40	95	41
97		2·49	70	25
99		4·42	123	28
101		3·85	107	23
103		4·08	113	33
105	3	3·40	95	35
107		2·95	82	28
109		4·65	129	23
111		3·85	107	30
113		2·77	77	23
115		3·27	91	29
117		3·18	88	21
119		2·61	73	22
121		3·63	101	32
123		3·29	91	31
125		3·46	96	27
127		3·18	88	38
129		4·20	117	21
131		3·63	101	33
133		3·63	101	32
135		3·32	90	40
137		4·31	120	24
139		3·54	99	23
141		3·52	98	33
143		3·97	110	27
145		4·65	129	32
147		4·25	118	24
149		2·95	82	23
151		3·40	95	39
153		4·42	123	37
155		5·02	139	31
157	4	3·15	88	25
159		3·74	104	29
161		3·40	95	25
163		4·14	115	32
165		3·35	93	25
167		3·88	108	29
169		3·18	88	24
171		4·54	126	32
173		3·74	104	34
175		3·63	101	27
177		3·63	101	35
179		3·63	101	26
181		3·40	95	24
183		3·91	108	34
185		4·03	111	27
187		3·63	101	39
189		3·40	95	30
190		3·18	88	39
191		3·86	107	30

TABLE I.—*continued.*

Absolute and Relative Birthweights of Sydney Infants of Different Orders of Birth and Ages of Mothers.—continued.

MALES.—*continued.*

Number.	Birth Order.	Weight.		Mother's Age.
		Kilograms.	Relative.	
192		3·86	107	42
193		4·08	113	32
194		3·40	95	26
195		4·31	119	32
196		4·76	132	30
197	5	4·20	117	34
199		3·86	107	37
201		3·86	107	31
203		3·63	101	37
205		2·95	82	31
207		4·42	123	27
209		4·42	123	24
211		3·86	107	37
213		4·08	113	35
215		4·88	135	34
217		3·63	101	40
218		2·61	73	35
219		3·86	107	34
220		3·91	109	30
221		4·54	126	31
222		4·88	135	41
223		3·52	98	31
224		3·23	89	30
225		3·52	98	29
226		4·31	119	31
227		4·42	123	46
228		2·95	82	26
229	6	4·42	123	29
230		3·52	97	38
231		3·97	112	37
232		3·29	91	36
233		3·63	101	32
234		3·40	95	27
235		3·97	110	33
245	7	4·31	120	39
246		4·54	126	36
247		3·63	101	36
248		3·97	110	30
274		4·08	113	39
259	8	3·66	102	31
260		4·54	126	41
262	9	3·63	101	30
263		3·63	101	34
264		3·91	109	39
267	10	4·31	120	41
269		2·95	82	36
271	13	2·61	73	37
273	14	3·29	91	38

FEMALES.

2	1	3·74	109	15
4		3·06	83	26
6		3·18	93	20
8		3·18	93	29
10		3·35	98	25
12		3·63	106	28
14		3·18	93	17
16		3·40	99	21
18		3·06	91	19
20		3·71	108	18
22		3·18	93	23
24		3·74	109	20
26		3·97	115	17
28		3·63	106	18
30		2·27	68	24
32		3·86	112	33
34		3·63	106	22
36		3·97	115	25
38		4·31	124	20
40		3·63	106	32
42		3·74	109	25
44		3·18	93	19
46		4·08	118	20
48		3·40	99	18
50		3·74	109	18
52		2·95	87	27
54	2	3·40	99	22
56		2·95	87	26
58		3·23	95	23
60		4·11	119	39
62		3·52	102	21
64		3·52	102	22
66		3·40	99	31
68		3·18	93	26
70		3·52	102	19
72		3·97	115	29
74		3·06	90	29
76		3·52	102	20
78		4·00	116	21
80		3·12	92	21
82		2·95	87	17
84		3·40	99	29
86		3·27	96	26

TABLE I.—*continued.*

Absolute and Relative Birthweights of Sydney Infants of Different Orders of Birth and Ages of Mothers.—continued.

FEMALES.—*continued.*

Number.	Birth Order.	Weight.		Mother's Age.
		Kilograms.	Relative.	
88		2·95	87	29
90		4·42	127	21
92		4·54	130	22
94		3·63	106	20
96		3·06	90	35
98		3·52	102	28
100		3·69	107	20
102		3·18	93	23
104		3·40	99	22
106	3	3·63	106	39
108		2·89	84	29
110		3·69	107	38
112		3·06	90	39
114		2·89	84	33
116		3·52	102	36
118		3·06	90	31
120		2·95	87	31
122		3·63	106	22
124		4·54	130	29
126		4·08	118	34
128		2·95	87	28
130		3·91	113	23
132		3·97	115	24
134		3·06	90	31
136		4·08	118	28
138		4·14	120	32
140		4·08	118	35
142		2·95	87	22
144		2·95	87	36
146		3·74	109	26
148		3·63	106	21
150		3·06	90	29
152		3·12	92	35
154		2·72	81	21
156		3·74	109	26
158	4	3·74	109	33
160		3·63	106	37
162		3·52	102	24
164		3·86	112	37
166		4·20	121	25
168		4·08	118	25
170		3·74	109	30
172		4·08	115	30
174		4·20	121	30
176		4·31	124	36
178		2·95	87	22
180		3·40	99	33
182		3·40	99	25
184		3·63	106	30
186		3·40	99	36
188		4·20	115	24
198	5	3·46	101	33
200		3·63	106	30
202		4·31	126	40
204		3·74	109	21
206		3·86	112	26
208		4·99	145	30
210		3·20	94	39
212		4·54	130	32
214		4·28	125	39
216		3·97	115	28
236	6	4·99	145	36
237		3·49	102	33
238		3·86	112	41
239		3·97	115	32
240		3·86	112	28
241		2·95	97	36
242		4·08	118	39
243		4·42	127	40
244		4·31	124	32
249	7	3·63	106	28
250		2·72	81	33
251		2·72	81	46
252		3·74	109	29
253		3·80	110	41
254		3·52	102	39
255		3·29	96	29
256		3·97	115	39
257		3·97	115	37
258		2·95	87	32
261	8	4·48	129	31
265	9	4·99	145	32
266		3·69	107	33
269	10	3·18	93	42
270		3·18	93	34
272	13	4·20	121	38

The figures for the male and female infants have been arranged separately in the table. To facilitate comparison between the two sexes, and also between the weights of infants at birth with the weights of older infants, these weights have been expressed as percentages of certain standard values. The figures taken as standard are those calculated by Brailsford Robertson (1915) for South Australian infants (see Table 5). The order of birth of the infants and the ages of their mothers are also given in Table I.

The only selection exercised in obtaining these weights for examination was to exclude all cases in which the histories gave evidence of any abnormality of either mother or child, and to take approximately equal numbers of infants of the different orders of birth. The latter requirement limited the numbers available, as later children are much less numerous than earlier children. In any series not selected in this respect, the group of first children will be considerably larger than the other group. The mean relative weight of the whole series is 103·5% of Robertson's figures. The differences are probably due in part at least to the fact that Robertson's figures are not selected with regard to order of birth and must have contained a larger proportion of earlier, relatively lighter infants.

When the relative weights of the infants of the two sexes are calculated separately, the values obtained are 102·2 for the males and 105·0 for the females.

The relation of the mean age of the mothers to the numbers of children which they had borne is shown in Table II.

TABLE II.

Mean Ages of Mothers of Different Numbers of Children.

Number of Children.	Mean Age of Mother.	Number Observed.	
		Males.	Females.
	Yrs.		
1	22·3	26	26
2	23·7	26	26
3	29·7	26	26
4	31·8	24	16
5	32·8	21	10
6 and over	35·3	21	24

The ages of the mothers increase, as they must, with the number of their children. The increase of mean age between the second and third children is, however, much greater than that between other consecutive children. This difference is rather striking, but it is not proposed to discuss in this paper the possible significance of the difference. It also appears when the infants of the two sexes are considered separately.

When the average order of birth of these infants is calculated for different relative weights, the figures shown in Table III are obtained.

TABLE III.

Relation of Mean Birthweight to Mean Order of Birth.

Birthweight.		Birth Order. (Mean.)	Number.
Absolute. (Range.) Kilograms.	Relative. (Mean.)		
2·26–3·16	75·6	2·92	48
3·18–3·62	90·3	3·13	73
3·64–4·07	103·4	3·73	93
4·09–4·52	116·0	4·33	41
4·54–4·98	131·1	4·70	20

This relation between mean relative birth weight and mean order of birth is shown graphically in Figure I.

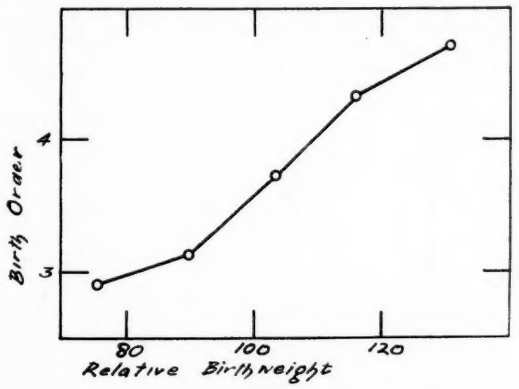

FIGURE I.
Mean birth weight (abscissæ) and mean birth order (ordinates).

The increasing proportion of later children with increase of relative weight is obvious from these figures. It is not safe, however, to draw conclusions from differences between the mean values of groups of results alone. It is true that here one is dealing, not with a difference between the mean values of two groups, but with four such differences, all of which fall consistently in the same direction. The probability that this consistent series of differences is due to chance is therefore likely to be small. As the orders of birth of the infants do not form a continuous series, but increase by steps, a correlation between the two series of values cannot be shown in the usual way.

The validity of the differences between the relative weights of infants of different orders of birth may, however, be shown in another way by determining the distribution about some arbitrary value of the relative weights of infants of different orders of birth. The standard deviation (σ) of this distribution is $\sigma = \sqrt{pq/n}$, where p and q are the proportions of the results above and below the arbitrary value respectively, and n the number of results in the group. The standard deviation of the difference between the distributions of two groups will be the square root of the sum of the squares of the standard deviations of the two distributions. The ratio of this difference to its standard deviation is a measure of the reliability of the difference, or of the probability against its being due to chance. The probabilities corresponding to various ratios of these differences to their standard deviations may be obtained from suitable tables, such as that in the useful article by Dunn (1929), in which the whole of this method of calculation is simply explained.

For examination in this manner the figures in Table I have been divided into three groups, as a finer subdivision gives groups containing rather too small numbers of results for this method of treatment. The arbitrary value of relative weight about which distributions have been calculated, has been taken as 99·8, the mean value for the first group. The distributions about this value, and the validity of the difference between these groups are shown in Table IV.

TABLE IV.
Dependence of Mean Relative Birthweights of Normal Infants on Order of Birth.

Birth Order.	Number.	Mean Relative Weight.	Number Above 99·8.	Odds Against Difference from Group I being due to Chance.
1–2	104	99·8	49	
3–4	92	101·6	52	5 to 1
5 and over	79	109·5	58	500 to 1

The figures in the table show that the chances are about five to one against the difference between the first and second groups being due to the hazards of sampling. The chances are 500 to 1 against the difference between the first and third groups being due to the same cause. The chances against both the second and third groups being greater than the first as a result of the hazards of sampling is 2,500 to 1. There is thus no reasonable doubt that these figures show a real increase in the birth weight of infants with the order of their birth.

Relative Weights of Breast-Fed Infants After Birth.

The relative weights of infants at various ages after birth have been calculated with reference to Brailsford Robertson's figures as described for the birth weights. Robertson's figures, expressed as metric units, are given in Table V for the range of age considered here.

TABLE V.
Weights of South Australian Infants (Brailsford Robertson).

Age in Weeks.	Weight in Kilograms.	
	Males.	Females.
0	3·60	3·44
4	4·42	3·99
8	5·22	4·76
12	5·85	5·31
16	6·53	5·94
20	7·21	6·53
24	7·76	7·08
28	8·17	7·58

In Table VI the relative weights, calculated as described, have been given for 145 normal, entirely breast-fed children. As in Table I, the figures are arranged according to order of birth. Within each group the figures run in order of relative weights. In addition, certain information with regard to the quantities of milk and its constituents in specimens obtained from the mothers is given. These figures will be discussed later.

In Table VII the mean orders of birth of these infants corresponding to different relative weights have been calculated as in Table III for the birth weights. In this case, however, each mean relative weight is calculated for a definite range of relative

TABLE VI.

Relative Weights of Infants, Physical Characters of Mothers, and Quantities of Milk Constituents Secreted by One Breast in Four Hours.

Number.	Sex.	Birth Order.	Relative Weight.	Weight in Grammes.		Calories.
				Fat.	Solids not Fat.	
9	M.	1	65	0·89	3·49	22·0
6	M.		67	0·91	1·76	15·2
8	M.		67	1·22	2·58	21·3
46	F.		74	1·44	2·08	21·0
11	M.		77	2·54	4·54	41·2
26	M.		78	3·17	8·75	63·5
142	M.		78	0·42	1·33	9·1
27	F.		80	2·65	3·61	38·2
134	M.		81	0·96	1·44	12·0
30	M.		83	2·32	3·22	3·38
120	F.		85	3·22	7·04	57·1
12	M.		85	1·87	3·40	30·5
153	F.		85	1·30	4·23	28·6
28	M.		86	2·59	3·45	37·2
54	M.		86	2·80	4·60	43·6
62	F.		86	2·27	4·95	40·2
141	M.		88	2·20	4·44	37·6
113	F.		88	4·45	7·71	70·9
72	M.		89	3·04	2·85	38·8
3	M.		91	1·76	3·09	28·2
5	M.		91	3·77	5·35	55·4
127	F.		91	2·65	3·98	49·7
57	F.		91	2·19	3·30	32·9
154	F.		91	4·12	5·26	58·2
31	M.		91	3·04	3·26	30·4
7	F.		92	2·95	5·85	50·0
18	M.		93	4·08	9·06	73·1
147	M.		93	2·42	4·05	47·0
92	F.		94	1·16	3·03	22·5
159	M.		94	4·14	8·97	73·0
4	M.		96	3·12	7·47	57·8
125	M.		96	5·28	7·08	75·8
109	F.		96	2·42	4·62	40·2
128	F.		96	3·74	6·31	58·9
13	M.		96	1·75	4·42	33·5
85	F.		97	4·25	6·48	64·0
23	M.		98	4·50	7·62	70·9
94	M.		98	2·17	3·38	33·0
14	M.		99	3·34	4·07	47·2
115	F.		99	3·25	5·85	52·6
53	M.		100	2·55	8·78	58·0
65	F.		100	3·50	7·02	59·6
108	M.		100	2·36	2·55	21·4
55	F.		102	1·95	3·09	30·0
157	M.		102	4·89	4·29	61·2
116	M.		102	2·94	3·53	40·5
52	F.		102	3·09	5·29	48·9
36	F.		103	3·18	6·95	56·4
148	M.		103	2·94	6·65	53·0
112	M.		104	3·58	5·87	55·7
39	M.		104	2·22	7·18	46·7
67	M.		105	6·46	14·10	113·9
69	F.		105	4·04	6·54	52·5
145	M.		106	1·26	2·07	19·7
10	F.		106	4·47	7·60	70·6
100	M.		106	6·65	17·20	128·9
161	F.		108	3·40	6·00	54·6
140	F.		110	1·60	4·73	33·3
59	F.		111	1·88	2·40	26·5
50	M.		111	2·06	1·64	25·1
146	F.		112	2·02	3·05	30·4
29	F.		113	3·66	5·68	55·7
66	M.		113	4·71	5·73	65·5
49	F.		116	5·65	8·54	85·0
162	M.		116	3·75	9·93	73·5
64	F.		119	7·56	14·53	126·1
77	F.		123	7·94	12·85	122·3
40	M.		123	2·23	5·20	41·3
25	F.	2	67	2·21	3·93	35·6
16	M.		75	1·49	2·25	22·4
82	M.		81	2·08	2·96	30·5
60	M.		82	4·46	7·96	72·0
144	F.		84	1·81	2·84	27·7
155	M.		84	1·90	3·15	29·7
78	F.		85	2·41	6·14	46·2
143	M.		85	0·94	1·39	14·1
2	M.		86	1·29	2·11	20·1
163	M.		88	1·89	4·85	35·4
79	M.		90	4·28	8·84	73·9
130	M.		90	1·60	3·70	29·2
97	F.		93	4·22	4·95	57·8
99	M.		95	5·04	8·82	80·7
56	M.		99	1·02	1·34	14·6
114	F.		100	1·75	3·95	31·6
124	M.		101	5·05	11·50	91·5
104	M.		103	5·32	12·35	97·1
41	F.		103	0·34	0·86	6·5
129	M.		105	1·63	3·90	30·3
98	M.		106	1·60	3·35	27·8
160	F.		110	2·13	4·66	37·8
89	M.		111	2·58	4·58	41·5
51	F.		111	2·09	4·72	37·7
158	M.		112	2·99	4·22	43·7
70	F.		114	2·33	2·45	30·8
68	F.		115	2·50	3·69	37·2
75	M.		115	2·90	5·68	48·8

TABLE VI.—*continued.*

Relative Weights of Infants, Physical Characters of Mothers, and Quantities of Milk Constituents Secreted by One Breast in Four Hours—continued.

Number.	Sex.	Birth Order.	Relative Weight.	Weight in Grammes.		Calories.
				Fat.	Solids not Fat.	
164	F.		117	1·83	5·20	37·3
156	F.		117	1·31	3·12	24·3
83	F.		122	2·87	6·32	51·1
61	M.	3	65	3·76	5·44	54·8
87	M.		77	1·86	3·10	29·2
20	M.		78	1·78	3·65	30·6
63	F.		93	1·82	7·52	46·6
96	M.		94	1·86	4·47	34·7
90	F.		100	2·05	6·12	43·0
76	F.		101	3·42	4·68	49·5
73	F.		104	1·75	4·54	33·9
95	M.		110	2·05	4·25	35·5
74	F.		114	5·17	11·59	92·9
43	F.		116	5·01	8·60	79·5
122	F.		121	4·52	6·32	66·0
33	F.		123	5·74	9·27	88·6
102	F.		124	7·15	11·80	111·7
48	M.		129	4·47	6·02	64·3
101	F.	4	83	1·66	2·64	25·5
152	F.		90	0·89	2·62	18·4
37	F.		98	3·50	5·44	53·4
123	M.		98	3·06	4·74	46·5
86	F.		99	2·18	2·89	31·2
44	F.		101	2·27	3·62	34·9
117	M.		104	1·58	4·58	32·3
107	M.		101	3·37	8·53	64·5
110	M.		108	6·95	9·79	101·5
15	F.		113	4·68	6·23	67·1
24	F.		113	1·11	2·62	20·4
131	F.		122	2·59	9·11	59·8
137	M.		124	5·38	8·55	82·7
106	M.	5	92	2·69	4·32	41·4
71	F.		99	2·32	7·42	50·7
103	M.		99	1·79	4·32	33·5
81	M.		105	2·58	8·23	56·2
150	M.		107	3·36	6·30	55·5
105	M.		110	4·44	7·48	69·9
32	M.		111	2·40	6·03	45·7
151	M.	6	94	3·63	6·12	57·2
80	M.		98	2·20	3·18	32·5
1	F.		111	3·30	7·04	57·9
149	M.		114	1·67	3·77	30·1
47	M.		128	5·02	10·85	89·0
118	M.		129	3·65	8·80	68·1
93	M.	7	99	2·76	2·46	34·6
88	F.		120	4·07	8·04	68·8
34	M.	8	96	1·35	3·90	27·9
133	F.		104	2·31	4·82	40·1
22	M.		116	2·56	8·67	58·1

TABLE VII.

Relation of Weights after Birth to Order of Birth.

Relative Weight.		Order of Birth. (Mean.)	Number.
Range.	Mean.		
80 and under	72·3	1·53	12
81–90	84·8	1·83	21
91–100	94·9	2·06	39
101–110	103·4	2·43	33
111–120	113·6	2·64	29
121–130	124·0	3·19	11

weights instead of for a range of absolute weights, as in Table III. These relations are shown graphically in Figure II.

The figures in Table VI show the same progressive increase in the proportions of later children with increasing relative weight as was seen for birth weights. Here again the caution observed with regard to the figures in Table II must not be lost sight of, although the probability of this succession of increasing values being due to chance must be small.

The figures for the relative weights have been divided into the same three groups as in Table III,

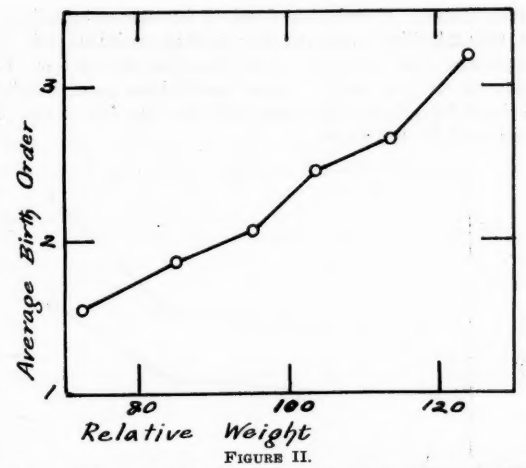

FIGURE II.

Mean relative weight (abscissæ) and mean order of birth (ordinates) of breast-fed infants of various ages.

and the probabilities of the differences between the first and the second, and first and third groups being due to chance have been calculated as already described. The figures are given in Table VIII.

TABLE VIII.

Dependence of Mean Relative Weights of Breast Fed Infants of Various Ages on Order of Birth.

Birth Order.	Number.	Mean Relative Weight.	Number Above 96·7.	Odds against Difference from Group I being due to Chance.
1–2	99	96·7	50	
3–4	28	104·1	21	111 to 1
5 and over	18	107·1	15	1000 to 1

The figures in Table VIII show that there are about 100 chances to 1 against the difference between the distributions of the relative weights in the first and second groups about an arbitrary value being due to chance. The arbitrary value about which the distributions have been observed is 96·7, the mean value of the relative weights of the first group. The chances against the similar difference between the first and the third groups being due to chance is 1,000 to 1. Here again there can be no reasonable doubt that the relative weights of the infants observed up to an age of about six months increase with the order of their birth. The higher values of the probabilities against these results being due to chance are, no doubt, due to the greater accuracy with which the infants in this group were weighed. The weighings were correct to the nearest half ounce (approximately 15 grammes). The possible error of the weights is thus only one-fourth that of the birth weights.

It will be noticed that the mean order of birth for each weight group is materially higher for the infants measured at birth (Table II) than for the older infants (Table VII). This difference is due to the fact, already mentioned, that the birth weights of approximately equal numbers of infants in the different orders of birth have been dealt with. No such selection has been exercised with respect to the older infants. A larger proportion of infants in the earlier orders of birth therefore occurs among them.

Growth of First and Later Infants.

A comparison of the weights at birth of the infants of different orders of birth with the weights of the older infants in the same orders indicates that the children tend to maintain the relative weights with which they are born. There is nothing to indicate that the later infants may grow faster than the earlier, within the period over which this examination extends. It would seem rather that, within this period, factors which led to the development of a heavier child during gestation continue in operation after the child is born. It is true that the initially heavier children must add to their bodies in a given time absolutely greater weights of material if they are to maintain their relative weights. Their relative weights are maintained, however, by the addition to their bodies in a given time of a weight of material which bears the same ratio to their original weight as does that added by the initially lighter infants.

These observations are not at variance with those of Mackinlay (*loco citato*). The more rapid growth of the first child which he observed did not make itself evident until towards the end of the first year of growth. The oldest infants in the present series were only six months of age, so that it must also be borne in mind that the present series comprises only the normal, healthy, and entirely breast-fed infants of healthy mothers. Even assuming that Mackinlay's infants were all normal, one cannot assume that they were all breast fed. They were, for the most part, the children of Glasgow parents in very indigent circumstances. Many of the mothers were in receipt of relief in the form of rations. In such families each additional child results in a seriously increased drain on already overtaxed resources, and there is every reason to suppose that the first children would have advantages of care and feeding not available to the later children. Differences of this kind in the provision for first and later children would not be so important with families in easier circumstances.

The figures recorded in Table IX give some idea of the relative growth of first and later infants of the present series.

TABLE IX.

Relative Weights of First and Later Children at Different Ages.

Age Group. (Weeks.)	Mean Relative Weight.		Number.		Above 97·7.		Odds against Difference being due to Chance.
	First.	Later.	First.	Later.	First.	Later.	
0	97·7	104·5	52	223	25	85	14 to 1
0–8	88·1	100·9	17	29	6	19	42 to 1
8–16	97·2	102·3	29	26	13	16	18 to 1
16–24	98·6	103·5	16	18	9	7	6 to 1
24 +	104·1	112·7	7	3	4	3	1 to 1

In Table IX the relative weights of first and later children in different age groups are recorded. Each age group comprises a period of eight weeks. The distribution about an arbitrary value of the figures in each group are also given. The value chosen is the mean value of the relative weights of the infants of *primiparæ* of the first age group, 97·7. From these distributions the probability that the difference between the mean relative weights of first and later children is not due to chance has been calculated as described. It will be seen from the table that from the second age group on the odds against the mean weights of the later children being greater than those of the first children as a result of the hazards of sampling become steadily less. No significance can be attached to the differences between the last two groups, when considered alone. The evidence supports Mackinlay's conclusion that the differences of relative weight between first and later children tend to become smaller as the children increase in age.

Even in the first three groups the odds against the direction of the differences between the first and later children being due to chance are not very high. When the odds against the differences between all three of these differences falling in the same direction as a result of chance are, of course, much greater, and amount to about 1,000 to 1. There is little doubt, therefore, that first children are definitely lighter than later children of the same age up to the sixteenth week.

It will be noticed that the difference between the relative weights of the infants in the second age group (0-8 weeks) is much greater than the differences between the other groups. The significance of this difference is also much greater than that of the other differences. Although the data hardly warrant a definite conclusion on the matter, it is tempting to speculate whether the growth of the first children may not really be more retarded during the first month or two of life than that of the later children. This seems by no means an impossibility when the greater liability to injury at birth to which first children are subject is considered. It would appear that first children, the children of *primiparæ*, start life with an even greater handicap with regard to birth weight than is indicated by their lower birth weights. Their subsequent overtaking of the later children is thus rendered all the more striking.

The figures shown in Table VIII have been plotted graphically in Figure III.

Relation of Weight of Breast-Fed Infants to Composition of Their Mothers' Milk.

The dependence of the weight of a young animal at a given age upon its intake of food becomes sufficiently obvious when this intake is varied between sufficiently wide limits. Between extremes, however, there is a range, both of food intake and of body weight, which must be regarded as normal. It has repeatedly been shown for laboratory animals that within this range the gain in weight for a given intake of suitable food of known composition is remarkably constant for young animals of the same age and species. It is possible to express the gain in weight under these conditions as a mathematical function of the food intake (see, for example, Jull and Titus, 1928).

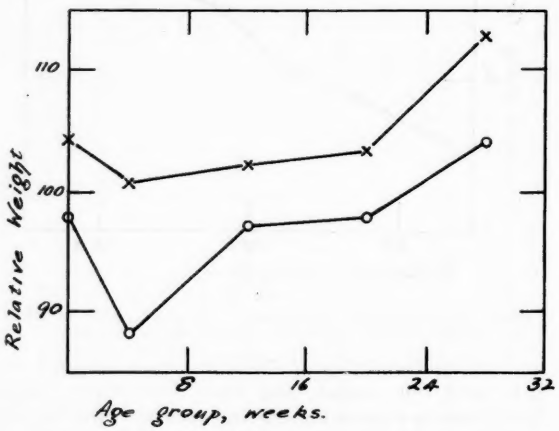

FIGURE III.
Mean relative weights of first (o) and later (x) breast-fed infants (ordinates) at various ages (abscissæ).

Observations of this kind have of necessity been carried out upon animals the diet of which was accurately controlled, both in quantity and composition. It is probable that similar regular relations exist between the food intake of suckling animals and the quantity and composition of the milk which they receive. Under these conditions, however, adequate control of the food supply is much more difficult, and in the human subject is even more difficult than with laboratory animals. The data on the composition and quantity of the milk obtained from the breasts of women after a known period of rest and the weights of their breast-fed infants obtained by Wardlaw and Dart (*loco citato*) afford material with which an attempt may be made to find some relation between their weights and some of the characters of their food supply. Such a relation may best be shown by calculating the correlation coefficients of the relative weights of the infants and the properties of the milk in question.

In Table X the correlation coefficients between the relative weights of the infants and the following quantities are given: (1) volume of milk obtained in four hours, (2) weight of fat, (3) weight of solids not fat, (4) weight of lactose, (5) weight of protein, (6) weight of ash, and (7) calorific value of the four-hour specimens. The coefficients for volume fat and solids not fat are calculated on 144 analyses, the figures for the remaining quantities on 120 analyses. In calculating the coefficients the assumption is implied that the four-hour specimens of milk from one breast are representative of the twenty-four-hour production of milk of both breasts. It has already been shown that there is no significant difference between the volume or composition of the

milk produced by each breast (Wardlaw, 1915). Whether the four-hour specimens are truly representative of the twenty-four hours' secretion is more open to question. The various specimens are, however, comparable. More serious is the probability that the periods of rest were very inaccurately measured, as the subjects themselves had to be relied upon to observe this period of rest of the mammary gland from which the specimen was obtained. Further, although every endeavour was made to empty the glands completely, it is unlikely that exactly the same proportion of its total content of milk was obtained in each instance.

TABLE X.

Correlation between Relative Weights of Breast Fed Infants and the Quantities of Milk and its Constituents obtained from One of their Mother's Breasts after a Period of Rest of Four Hours.

Observation.	Coefficient of Correlation.	Standard Deviation of Coefficient.
Volume of milk	+0·460	±0·048
Weight of fat..	+0·456	±0·031
Weight of solids not fat	+0·382	±0·081
Weight of lactose	+0·256	±0·058
Weight of lactose	+0·408	±0·052
Weight of protein	+0·400	±0·053
Calories	+0·387	±0·048
Weight of ash	+0·408	±0·052

In spite of the errors of sampling to which the specimens were subject, Table X shows that definite positive correlations exist between the weights of of the infants at given ages and the volumes of milk obtained, and the weights of the various materials contained in the specimens. The degree of correlation shown in the table is not high, but the low values of the standard deviations indicate that the correlations are quite significant. The conclusion is then justified that relatively heavier breast-fed infants have available a more plentiful food supply than relatively lighter infants.

Having demonstrated the existence of a definite correlation between relative weight and food supply, it may be permissible to show this relation graphically by plotting mean values of quantities of milk and its constituents against mean values of relative weights. In Table XI the mean volumes of milk, weights of fat, and weights of solids not fat are given which correspond with the mean values of the relative weight of weight groups increasing by 10 of the standard value.

TABLE XI.

Mean Relative Weights of Breast Fed Infants and Corresponding Mean Quantities of Milk and its Constituents obtained from a Breast after a Rest Period of Four Hours.

Mean Relative Weight	Mean Volume of Milk (c.cm.).	Mean Weight of Fat (grammes).	Mean Weight of Solids not Fat (grammes)
72·3	39·9	1·81	3·58
84·8	48·3	2·23	4·04
94·9	60·1	2·88	5·12
103·4	73·2	3·16	6·64
113·6	78·0	3·07	5·74
124·0	73·5	4·64	8·51

The general trend of the figures in Table XI are shown graphically in Figure IV.

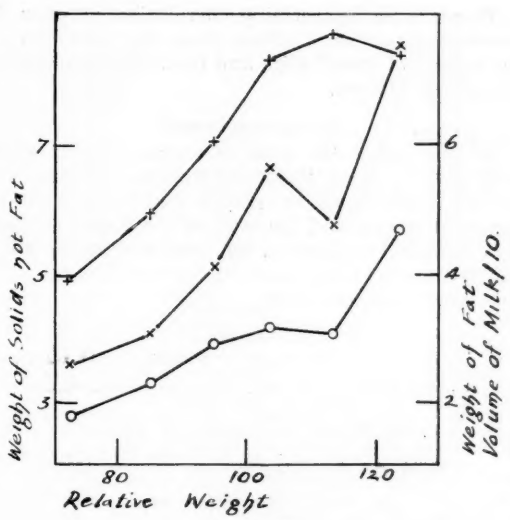

FIGURE IV.

Mean volumes of milk (+), weights of fat (o) and of solids not fat (x) obtained from a breast after a period of rest of four hours (ordinates), and relative weights of breast-fed infants (abscissæ).

When the concentration of the constituents of the samples of milk are considered in relation to the relative weights of the infants, a very different state of affairs is revealed. The coefficients of correlation between the relative weights of the infants and the percentage of fat and of solids not fat in the specimens of the milk which they receive are given in Table XII.

TABLE XII.

Correlation between Relative Weights of Breast Fed Infants and Concentrations of Constituents of their Mothers' Milk.

Observation.	Coefficient of Correlation.	Standard Deviation of Coefficient.
Percentage of fat	+0·047	±0·010
Percentage of solids not fat	+0·073	±0·010

The coefficients of correlation shown in Table XII are so low as to justify the conclusion that for all practical purposes the concentration of the milk consumed bears no relation to the relative weights of the infants consuming it.

Summary.

The earlier infants of a moderate sized family tend to be lighter than those born later.

For breast-fed infants a similar relation between weight and order of birth holds up to an age of about six months.

The growth of the infants of *primiparæ* tends to be retarded more during the first four weeks of life than the growth of later children.

The relatively heavier, later children of a family have available a more plentiful supply of breast milk than the earlier, lighter children.

There is no appreciable correlation between the concentration, as distinct from the quantity, of the supply of breast milk and the relative weights of breast-fed infants.

Acknowledgements.

In conclusion, we wish to express our indebtedness to Dr. Elma Sandford-Morgan and the staff of the Newtown Baby Health Centre, and to Dr. Bruce Williams and the staff of the Royal Hospital for Women, Paddington, for their assistance, which enabled us to obtain much of the material on which this work has been done.

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Reports of Cases.

NON-SUPPURATIVE INFLAMMATION OF THE ANTERIOR DEEP CERVICAL AND TONSILLAR LYMPH GLANDS FOLLOWING TONSILLECTOMY.

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THE complication herein reported is so unusual that it is thought advisable to place it on record.

Miss P.B., aged twenty-five years, had had three attacks of quinsy, the last being on the left side three months before operation. She had not had her tonsils operated on before; she had had measles and chicken pox as a child, and also mumps. Her teeth were artificial in the upper jaw and in good condition in the lower.

An ordinary routine dissection of the tonsils was done on March 19, 1932, with a Boyle Davis gag and ether anæsthesia. Nothing remarkable was noticed about the right tonsil, and nothing on the left side, except two or three very tough fibrous cord-like adhesions, which had to be divided. There was no more than the usual hæmorrhage, and she left the theatre with both fossæ dry. The sister in charge saw her the following morning at 7 a.m. and noticed nothing unusual, but at 12 noon, five hours later, noticed a large swelling in the left side of the neck. The patient complained now, for the first time, of severe pain in the region of the swelling, which had apparently come up suddenly. Examination revealed a tender, tense swelling, about the size of half a peach, in the region below the angle of the jaw, in the vicinity of the anterior group of deep cervical lymph glands and the so-called tonsillar gland, that is, the gland lying in the angle between the internal jugular and the common facial veins. This swelling faded away about five centimetres (two inches) below the angle of the jaw into the sterno-mastoid muscle. There were no signs of staining of the skin over the area, nor did any appear during the next two weeks. Her temperature was just a little raised. The temperature gradually subsided as the swelling went down during the next six days, when she had a small secondary hæmorrhage of about three to four ounces. The following morning the swelling had gone up again a little, and so had the temperature. During the next two weeks the temperature and the swelling gradually subsided and the neck became normal.

It is interesting to note that at no time did the area of the tonsillar fossa or pillars or palate look anything but as is expected following a tonsillectomy. Also that during the time the swelling was subsiding it remained hard, and at no time was there even a suspicion of fluctuation or of staining of the tissues, either inside or outside, with hæmoglobin, thus, we think, excluding a hæmorrhage into the tissues. Careful questioning now elicited the story that nine years ago, after she had her upper teeth extracted, she had, as she said, "an almost similar condition around the upper jaw".

PERNICIOUS ANÆMIA TREATED WITH LIVER EXTRACT GIVEN INTRAMUSCULARLY.

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PHYSICIANS abroad are hastening to report their early successes in the treatment of pernicious anæmia with liver extract administered parenterally, and confirm the experiments of Gänsslen and others which showed that this mode of administration remarkably enhances the potency of the active principle; but so far no case has been recorded in the medical literature of Australia to bring to the notice of practitioners that this new method of treatment is fully available in this country.

A woman, aged seventy-four years, was admitted to the Royal North Shore Hospital of Sydney on July 29, 1932. She had previously been treated in the hospital for pernicious anæmia, and Table I shows her condition and her response to treatment from 1928 to 1931.

Upon admission on this last occasion she was in a stage of profound relapse, very gravely ill. Her skin was lemon yellow; the conjunctivae were stained. She was orthopnœic. She vomited food and liver extract given by the mouth. The pulse was frequent and thready. There was some hypostatic pulmonary œdema. The urine contained a faint cloud of albumin, but no bile. The blood count was as follows: red cells 964,000 per cubic millimetre, hæmoglobin value 23%, colour index 1·2, leucocytes 1,700 per cubic millimetre. The red cells showed considerable irregularity of size, shape and staining; many macrocytes were present; a megaloblast was seen; platelets were scanty.

It was intended not to perform blood transfusion on account of its danger to these patients, but two days later the patient was so exhausted and obviously dying that it was undertaken with the utmost precautions against accident. However, when 260 cubic centimetres of a mixture of citrate solution and blood had been administered

TABLE I.

Date.	Red Cells per cubic millimetre.	Hæmoglobin Value.	Colour Index.	Leucocytes per cubic millimetre.	Type of Blood Picture.[1]	Clinical Notes.
May 7, 1928	1,830,000	35	1·0	3,125	1	Weakness, sore tongue, numbness of legs and cramps in the feet, "pins and needles" in hands, yellowish complexion, liver slightly enlarged, hæmic murmurs, complete achlorhydria, numerous microbes in the gastric juice.
May 21, 1928	1,560,000	32	1·0	3,750	2	Administration commenced of liver extract (Parke, Davis and Company), one vial three times a day.
May 25, 1928	1,690,000	34	1·0	4,375	2	
June 5, 1928	1,980,000	35	1·0	5,000	2	
June 15, 1928	2,380,000	40	0·9	5,625	1	Patient left hospital. Symptoms improved.
September 30, 1929	1,620,000	32	1·0	2,000	1	Abdominal pains, epigastric tenderness, diarrhœa, jaundice (biliary disease excluded), tingling and numbness of hands. Liver extract administered as before.
October 11, 1929 .	2,300,000	44	0·9	3,500	1	Transfusion of blood (450 cubic centimetres).
October 28, 1929 .	2,500,000	43	0·9	2,800	2	Much improvement subjectively.
November 14, 1929	2,200,000	40	0·9	3,000	3	Improvement maintained. Left hospital.
February 15, 1931	1,800,000	37	1·0	3,500	2	Taking "Karna Vita" and feeling well till six weeks previously. Jaundice, undue dyspnœa, paræsthesia, slight tenderness over spleen, liver not enlarged, crepitations at both bases. Liver extract administered, the equivalent of eight ounces of fresh liver daily.
February 22, 1931	1,910,000	38	1·0	3,625	2	
March 2, 1931 ...	2,300,000	42	1·1	4,000	2	
March 16, 1931 ...	2,800,000	58	1·0	6,250	4	Marked improvement in blood picture.
March 30, 1931 ...	2,990,000	60	1·0	5,000	4	
April 10, 1931 ...	3,100,000	60	1·0	6,250	4	Is very well. Discharged.

[1] Types of blood picture: 1 = red cells show marked irregularity in size, shape and staining, many macrocytes, an occasional megaloblast. 2 = the same, but no nucleated red cells. 3 = the same, an occasional normoblast. 4 = much improved.

TABLE II.

Date.	Red Cells per cubic millimetre.	Hæmoglobin Value.	Colour Index.	Leucocytes per cubic millimetre.	Type of Blood Picture.[1]
July 29, 1932	964,000	23	1·2	1,700	1
August 2, 1932	960,000	20			
August 5, 1932	1,100,000	22	1·0		
August 8, 1932	1,684,000	34	1·0		
August 12, 1932	1,987,000	40	1·1	4,000	1
August 18, 1932	2,800,000	67	1·2		
August 29, 1932	3,875,000	82	1·1		4
September 9, 1932	4,200,000	91	1·1		

[1] 1 = red cells show marked irregularity in size, shape and staining, many macrocytes, an occasional megaloblast. 4 = much improved.

the patient vomited and was collapsed for fifteen minutes. Two days later the red cell count was 960,000 per cubic millimetre. The dispenser was instructed to purchase some "Campolon", the "intramuscular liver extract" of Bayer Products Limited, and on August 2, 1932, the first injection of six cubic centimetres was given. Another dose of six cubic centimetres was given the next day, and a day later a course of two cubic centimetres given daily was commenced. (Each cubic centimetre of "Campolon" is said to contain the active principle derived from 2·5 grammes of raw liver.)

The response was immediate and remarkable, both in the clinical condition and in the state of the blood (shown in Table II). The dyspnoea became much less. Her strength increased. She retained food. The pulmonary œdema, which was becoming more marked, disappeared in a few days. The jaundice disappeared. The reticulocyte count rose to 19% by the sixth day. Now, after forty days of treatment with "intramuscular liver extract", this patient of seventy-four years is walking about, and her red cell count is nearly normal.

Acknowledgement.

Dr. W. Wilson Ingram, under whose care the patient was admitted to hospital, kindly permits us to report this case.

Reviews.

SEPTAL CAUTERIZATION IN ASTHMA.

"THE FRANCIS TREATMENT OF ASTHMA", by A. Francis, is not, as the author states, a text book on asthma, but an exposition of his method of treatment.[1] Let it be said at the outset that, whether or not we agree with his hypotneses, the publication is obviously the work of an honest observer whose results speak for themselves; and, as the treatment advocated by him for this distressing complaint is so simple and the results by other methods so unsatisfactory, it demands a universal and fair trial.

In his book Francis argues and brings definite evidence to show that asthma is due to a vaso-motor neurosis or instability, and the aim of his treatment is to stabilize the vaso-motor system. This stabilization is brought about by light cauterization of the nasal septum. His results by this simple method of treatment are remarkable and spectacular, as a long series of published cases show;

[1] "The Francis Treatment of Asthma", by Alexander Francis, M.B., B.Ch., B.A., M.R.C.S.; 1932. London: William Heinemann (Medical Books) Limited. Demy 8vo., pp. 159. Price: 7s. 6d. net.

practically 60% of all patients obtained complete relief. What other method of treatment can approach this either in its simplicity or effectiveness? At this stage it is interesting to note that W. N. Robertson, who was associated with Francis in some of his early cases, has obtained the same percentage of permanent relief by a similar method of treatment during many years of practice.

The type of asthmatic in whom a good result is nearly always assured, is the one with a good blood pressure and a normal nose; unfavourable patients are those with a very low blood pressure and nasal polypi. Francis sounds a note of warning against removing polypi from asthmatics, especially those with low blood pressure and anaphylactic to aspirin. On this point he is bound to meet with opposition, because, after all, the presence of nasal polypi indicates some form of nasal accessory sinus infection, and it is hard to believe that a patient can receive anything but benefit from having these efficiently removed, be he asthmatic or otherwise.

It is rather a pity that the actual technique of his nasal cauterization is not fully described, but he stresses that the cautery must be applied lightly, only the merest touch being necessary in most cases. No clear explanation is forthcoming as to how this nasal cauterization stabilizes the vaso-motor system, but in certain cases it definitely lowers the blood pressure and most of its good effects may be due to this fact. There is evidence to show that this effect on the blood pressure is due to vaso-motor action, through the sympathetic on the cutaneous vessels.

After all, results are what matter, and Francis has them in plenty. His book can confidently be recommended to all, especially oto-rhino-laryngologists, whose opportunities for thoroughly testing his method of treatment are greater than those of other practitioners.

BACTERIOLOGY.

THE publication of a third edition of the "Handbook of Bacteriology", by Professor J. W. Bigger, within seven years of the first edition is evidence of the need for such a book, and also a tribute to the manner in which this need has been met.[1] The work, which is primarily intended for students of medicine and for those practitioners who have not specialized in bacteriology, gives a clear and adequate account of the main bacteria affecting man.

In the latest edition alterations, particularly in the chapters on immunity, have been effected to bring this work up to date, although the size of the book has not been increased to any material extent.

This book can confidently be recommended to those for whom it has been written, as giving a short but accurate presentation of a very extensive subject in which advances are constantly being made.

WOMAN AT THE CLIMACTERIC.

"THE CRITICAL AGE OF WOMAN" is a small volume of one hundred and fifty odd pages. It is written mainly on woman at the time of the menopause and to a lesser extent on her whole sexual life. Its author is a man, Walter M. Gallichan, a layman.

There are eight chapters in all. The first three chapters deal with woman's sexual life, the next four with the menopause, and the last with woman in old age. The average person reading the book would be prone to get the idea that, morning, noon and night, most women, perhaps subconsciously, were wanting sexual intercourse or preparing or hoping for it. For example: "Nine women out of ten will vigorously deny the existence of a longing that is often almost obsessional." The writer explains that many of women's ways are due to sex impulse, as "I am inclined to associate the sex impulse with climacteric kleptomania".

To those who might be called upon to treat the ever-varying moods of woman the book will be useful, the contents may not be pleasant reading; but to understand and care for the female sex such possibilities must be borne in mind, as one authority has said, the menopause may turn "a devil into an angel and an angel into a devil".

[1] "Handbook of Bacteriology for Students and Practitioners of Medicine", by Joseph W. Bigger, M.D., Sc.D. (Dublin), F.R.C.P.I., D.P.H., M.R.I.A.; Second Edition; 1929. Baillière, Tindall and Cox. Post 8vo., pp. 468, with illustrations. Price: 12s. 6d. net.

[2] "The Critical Age of Woman", by W. M. Gallichan; 1932. London: Noel Douglas. Crown 8vo., pp. 160. Price: 4s. 6d. net.

Notes on Books, Current Journals and New Appliances.

A GERMAN-ENGLISH MEDICAL DICTIONARY.

MEDICAL practitioners who are finding it necessary to make an attempt to master the German language in order that they may read articles in medical journals will welcome the fourth edition of "Lang's German-English Dictionary of Terms Used in Medicine". This edition is larger than the third by 313 pages. It is clearly and attractively printed on good paper. There is no need to emphasize the value of this work; the third edition was well known, the fourth will enhance the reputation of the work.

A DICTIONARY FOR NURSES.

MISS LOIS OAKES has published "A New Dictionary for Nurses".[2] The author is described *inter alia* on the title page as "Examiner to the General Nursing Council of England and Wales". The most pleasing feature of this book is its appearance. The information contained within its attractive covers is on the whole satisfactory. Some of the definitions, however, are slovenly, some are too full, others are ungrammatical. "Cholecystography" is "examination of the gall-bladder after it is rendered opaque to the X-rays". "Virus" is "a disease-producing or poisonous substance; any protozoa or bacteria capable of causing disease". "Foreskin" is "the prepuce or skin covering the male urethra". "Cocaine (HCl), $\frac{1}{16}$ to $\frac{1}{4}$ gr" will convey nothing to a nurse; to a chemist it is nonsense. "Unction" is "to anoint". The amœba is described in detail and a line drawing of a distressful-looking amœba is reproduced. It is a pity that a nurse should try to produce a book of such pretensions.

THE WAR AND THE DEAD.

J. C. WATERS has written a book entitled: "Crosses of Sacrifice: The Story of the Empire's Million War Dead and Australia's 60,000."[3] It is dedicated "To the Diggers and to All Who are Kin to the Empire's Million War Dead". The author made a pilgrimage to the war cemeteries in Gallipoli, Egypt, Palestine, France and Flanders. He brought back with him pictures of war cemeteries and memorials, which are published in this book. He describes them and gives a short account of the actions which took place in each centre. This is a book which may be given to those of the rising generation who know nothing of the horrors of war and who find it difficult to realize the appalling wastage of human lives that took place.

[1] "Lang's German-English Medical Dictionary", revised and edited by Milton K. Meyers, M.D.; 1932. Philadelphia: P. Blakiston's Son and Company, Inc. Medium 8vo., pp. 934.

[2] "A New Dictionary for Nurses", by Lois Oakes, S.R.N., D.N.; 1932. Edinburgh: E. & S. Livingstone. Demy 18mo., pp. 362, with illustrations. Price 3s. net.

[3] "Crosses of Sacrifice: The Story of the Empire's Million War Dead and Australia's 60,000, by J. C. Waters, with a foreword by General Sir Harry Chauvel; 1932. Australia: Angus and Robertson, Limited. Crown 4to., pp. 130, with illustrations. Price: 6s. net.

The Medical Journal of Australia

SATURDAY, NOVEMBER 5, 1932.

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MATERNAL CARE.

In a recent issue reference was made to the final report of the Departmental Committee appointed in Great Britain in 1928 "to advise upon the application to maternal mortality and morbidity of the medical and surgical knowledge at present available, and to inquire into the needs and directions of further research work". On that occasion attention was directed to the general scope of the inquiry and to the more important conclusions. Other aspects of the report are of great interest, and among them is that of "the betterment of the provision for maternal care throughout the country".

The report deals solely with midwifery practice in Great Britain. It is therefore not applicable in all its details to Australian conditions. At the same time the essentials of a scheme for maternity service described by the Committee should be considered by Australian obstetricians. It should be pointed out that the essentials of a scheme for maternity service stated in this report are the same as those laid down in the *interim* report of 1930. The suggestions for an improved service made in the *interim* report were accepted by the Government of that time, and preliminary steps were taken to give them practical effect. Since then, however, the financial position of the country precluded substantial expenditure for this purpose. The essentials formulated by the Committee are as follows:

1. The provision in every case of the services of a registered midwife to act either as midwife or as maternity nurse.
2. The provision of a doctor to carry out ante-natal and post-natal examination in every case, and to attend, as may prove necessary, during pregnancy, labour and the puerperium, all cases showing any abnormality.
3. The provision of a consultant, when desired by the doctor in attendance, during pregnancy, labour and the puerperium.
4. The provision of hospital beds for such cases as need institutional care.
5. The provision of certain ancillary services.

Under the provisions of the acts in Great Britain dealing with midwifery and maternal and child welfare, many of these requirements are or may be met. Many of the services for which provision may be made are interesting and show the trend of legislation. The midwife's fee may be paid in necessitous cases. There is statutory obligation for the payment of a doctor's fee when he is called to the assistance of a midwife. There is also an obligation to compensate a midwife when she is suspended from practice on account of infection, if she is not in default. Midwives may be compensated for loss of patients sent to a maternity hospital from an ante-natal clinic. There is provision for "home helps". Provision is made for hospital accommodation, for ante-natal and post-natal supervision and for consultant and specialist services.

Many of the essentials laid down by the Committee are being put into practice in Australia, though not many of them as a result of parliamentary enactment. It will be seen that in Great Britain the tendency is to regard normal labour as the province of the midwife. This is the practice in some Continental countries. Whether such a practice would be best suited to Australian conditions is debatable; its acceptance, however, would imply that every woman prior to labour had received adequate ante-natal supervision. An increasing number of hospital beds are being made available throughout the Commonwealth for midwifery

patients, and we may conclude that no woman desirous of obtaining skilful treatment in her pregnancy will fail to get it. Although this is a satisfactory state of affairs, it is not sufficient. Nor will it suffice until every pregnant woman realizes the importance of skilful supervision from the time of conception till the end of the puerperium and until she determines to have it. The development of such an attitude of mind throughout the community will take time. The whole community is included, because the opinions of husband, relatives and friends carry much weight with a patient. The Departmental Committee states that the steady pressure of common sense, public opinion and fashion will eventually induce a patient to turn to her doctor for early and continued supervision because everyone takes it for granted that she should. The Committee puts the matter well:

> The best advertisement for ante-natal care, and one which will probably accomplish more than mere *ad hoc* propaganda, is the successful outcome of maternity cases that have received ante-natal supervision by midwife or doctor. On the other hand, an unfavourable outcome for mother or child may do very much to bring ante-natal care into undeserved disrepute. For these, among other reasons, it is essential that the ante-natal care should reach the highest possible standard of excellence and that no slipshod work should be allowed to pass muster by either midwives or doctors.

Ante-natal care is not the end of the matter. Post-natal care is equally important. The Departmental Committee expresses the view that it has so far received little attention. Its findings in regard to maternal morbidity would appear to justify this conclusion. Discussion of this aspect of the subject must be left for the present, but emphasis may be laid on the examination of the patient at the end of the puerperium, so that structural abnormalities consequent on delivery and incipient pathological conditions may be discovered. In conclusion, mention must be made of the "provision of home helps" referred to in the report. This should be thought of by the supervising practitioner as the pergnancy of his patient advances. A speaker at a Branch meeting some years ago, a medical woman, caused a little amusement, but talked common sense when she emphasized this point. She said that it was quite a common thing in her practice to see husbands doing the week's washing on Sundays in order to save their wives, who were in the latter months of pregnancy. She insisted that the husbands should do this and similar laborious household work. She was quite right in her contention that male practitioners should think of relief for the wife. Too much attention may be paid to methods and to new advances and not enough to the patient as a woman.

Current Comment.

PERNICIOUS ANÆMIA.

DURING the past few years Castle and his associates have carried out a great deal of investigation into the relationship between pernicious anæmia and abnormalities in the gastric secretion. They have come to the conclusion that the normal gastric juice contains a certain intrinsic factor which interacts in some way with an extrinsic factor in the food. The result of this interaction is the stimulation of hæmopoiesis. If their theory is correct, it follows that absence of either the intrinsic or extrinsic substance might cause anæmia. True pernicious anæmia is due to the absence of the intrinsic factor; anæmias simulating pernicious anæmia may be due to the absence of the extrinsic factor. The theory is an attractive one.

The nature of the hypothetical extrinsic substance has been discussed by Castle and his fellow investigators. They have described it as "protein or a closely related substance". In a more recent communication, by Maurice B. Strauss and William B. Castle, their views are modified somewhat.[1] They now conclude that the extrinsic factor is a substance closely related to vitamin B_2 (the antipellagra vitamin), if not vitamin B_2 itself. Spleen pulp digested with normal gastric juice was administered to two persons suffering from pernicious anæmia; the excellent response led Strauss and Castle to believe that the extrinsic factor was a nucleo-protein. When the experiment was repeated in three other cases, there was no reticulocyte response; it was concluded, therefore, that the extrinsic factor was not nuclear material, but some substance that occurred in variable quantities. For various reasons the observers' attention was directed to vitamin B_2. A preparation of autolysed yeast (containing vitamins B_1 and B_2, proteins and nucleo-proteins) was administered to a patient suffering from pernicious anæmia; it had no effect. But the administration of the same substance after it had been treated with normal human gastric juice was met with a rapid and great reticulocyte response. The preparation of autolysed yeast was subjected to heat by steam under pressure, with the object of destroying vitamin B_1. It is believed that at least half the vitamin B_2 is also destroyed by this

[1] *The Lancet*, July 16, 1932.

process. The administration of the modified substance, together with normal gastric juice, caused a prompt and characteristic reticulocyte response. Further tests were made with a view to investigating the possible effects of protein, nucleo-protein and nucleic acid. In only one instance, when nucleo-protein was given, did any response in the way of reticulocyte formation occur; in this instance it was thought that all the vitamin B_2 in the substance used in the test had not been destroyed.

Strauss and Castle suggest that an inexpensive substance for the treatment of pernicious anæmia may be prepared by the mixing of mucosa of hog's stomach (a source of the intrinsic factor) and a substance containing vitamin B_2; the mixture could be desiccated and powdered. They also suggest that the withholding of vitamin B_2 from the diet of laboratory animals may cause the development of a macrocytic anæmia resembling pernicious anæmia. The experimental production of pernicious anæmia would be of value as providing a means of testing the potency of liver extract. They point out that liver extract containing vitamin B_2 would have to be administered parenterally for testing purposes; for, if it were administered by mouth, the interaction between the vitamin and the intrinsic factor would mask the effects of the liver extract itself.

In view of the conclusions drawn by Strauss and Castle, the observations made by John F. Wilkinson on the gastric secretion in pernicious anæmia are of more than ordinary interest.[1] Wilkinson analysed the stomach contents of 208 persons suffering from pernicious anæmia and found that free hydrochloric acid was present in eight cases. He gives a brief history of each of these eight cases, as well as a record of the blood counts. The appearances were identical with those of pernicious anæmia; but the reader might be pardoned for scepticism concerning the diagnosis. The gastric secretion of one patient was incubated with beef muscle, according to the method described by Castle, and administered to another patient suffering from pernicious anæmia. The fact that no response in blood formation was evoked by this treatment is taken by Wilkinson to indicate the absence of the intrinsic factor from the first patient's gastric juice. There is a possibility that the beef muscle was lacking in the extrinsic factor. Interesting information might have been obtained if a preparation of vitamin B_2, without normal gastric juice, had been given to Wilkinson's eight patients.

The work carried out by Strauss and Castle marks another step towards solution of the problem of the macrocytic anæmias. Medical practitioners, especially those in tropical countries, should examine the diet of persons suffering from anæmia; they should apply the therapeutic test of administering vitamin B_2, if the diet is lacking in this constituent. A great deal of careful observation must be carried out in the laboratory and at the bedside before the views held by Strauss and Castle can be accepted. Even if they are eventually proved correct, they cannot be taken as telling the whole story of the complex ætiology of macrocytic anæmia.

[1] *The Quarterly Journal of Medicine*, July, 1932.

HEAD INJURY.

The importance of the diagnosis of fracture of the skull and of intracranial damage in head injury is sufficient justification for drawing attention to an analysis of the findings in one thousand patients admitted to Bellevue Hospital, New York, with a clinical diagnosis of fracture of the skull.[1] S. B. Wortis and F. Kennedy, who make the analysis, tabulate the findings and the results of treatment and draw some general conclusions. They classify head trauma clinically according to the pathological changes. They give four main groups: (i) simple scalp wounds, (ii) skull fracture with or without associated brain injury, (iii) brain injury with or without associated skull injury, (iv) combinations of the first three groups. The cases included in the review are confined to those in which: (i) *post mortem* evidence of skull fracture or brain laceration was present, (ii) X ray evidence of fracture of the skull was present, (iii) blood-stained fluid was obtained on lumbar puncture. It is interesting to note that in only 499 of the 1,000 cases was an X ray examination of the skull made, and in 230 of these no fracture was found. These figures indicate the fallaciousness of relying on clinical appearances. Further, Wortis and Kennedy point out that skiagrams should always be taken through at least two different diameters. Of the thousand patients 378 died, and autopsy was conducted in 257 instances; in 91·8% of these laceration of the brain was found, and in 59·1% hæmorrhage of the brain. In 846 cases lumbar puncture was done and record was kept of the findings in the cerebro-spinal fluid. The findings are tabulated and it is clear that the longer the period of unconsciousness, the poorer is the prognosis as regards life. Of the 791 patients whose cerebro-spinal fluid was blood-stained, 292 lived and 499 died. Many patients (154) died before lumbar puncture could be performed. The importance of lumbar puncture in head injuries was discussed in these pages in March of this year. The most important practical point in this paper is the reported success of conservative treatment. Surgical operation was performed in 37 cases only. One of the operations was trephining and 36 were subtemporal decompression. The treatment most usually adopted was that directed towards the reduction of intracerebral pressure—repeated lumbar puncture, the use of sulphate of magnesia, the use of caffeine and of a hypertonic solution of glucose. The main object of Wortis and Kennedy has been to demonstrate the success of conservative treatment. What needs to be remembered is that conservative treatment does not mean masterly inactivity, a refraining from operation and nothing more.

[1] *Surgery, Gynecology and Obstetrics*, September, 1932.

Abstracts from Current Medical Literature.

MORBID ANATOMY.

Tuberculosis of Arteries.

H. L. SHEEHAN AND R. WHITEHEAD (*The Journal of Pathology and Bacteriology*, September, 1932) report two cases of arterial tuberculosis. If these two cases are included, thirty-one cases of aneurysm and rupture of tuberculous systemic arteries have been reported. In the first of the authors' cases tuberculosis affected the right subclavian artery; in the second case a branch of the splenic artery was affected. In neither instance was there evidence of generalized tuberculosis. The invasion of the arteries probably occurred from without. In the first case the artery was probably infected from an adjacent lymphatic gland. From the fact that the artery was not dilated it is concluded that its wall was eroded rapidly. In the second case the tuberculous focus was in the splenic tissue and a false aneurysm had formed. The authors describe the histological appearances in detail.

Experimental Nephritis.

H. L. SHEEHAN (*The Journal of Pathology and Bacteriology*, July, 1932) reports investigations in experimental nephritis. He has injected rabbits with styryl quinoline compound number 90; this substance is 2(*p*-acetylamino styryl) 6-dimethylaminoquinoline methochloride. Injection of this substance produced an acute nephritis, characterized by necrosis of entire first convoluted tubules, chiefly in the outer parts of the cortex; this necrosis was followed by regeneration of the tubular epithelium. After about nine days a fibrosis develops around the regenerated tubules, which gradually atrophy and disappear. The glomeruli remain intact in the resultant scars for about six months and then become fibrosed by a process similar to ischæmic atrophy. In those animals which lived for over two months after the production of a moderately severe nephritis, the blood urea was permanently slightly raised, but the concentrating power of the kidneys became normal. The author points out that not much is known of the way in which specific effects are produced on the kidney by substances which give rise to acute tubular nephritis. He refers to the studies of many authors, and adds that it appears from them that certain tubular nephrotoxins, and possibly all, are accumulated in the kidney and thus produce their deleterious effects. The question to be determined is the fate of this nephrotoxin after its administration to an animal. When a harmless dose of the dye is given, it is taken up directly from the blood stream by the epithelium of the first convoluted tubules and of the broad ascending limbs of Henle. This absorption depends on the physiological activity of the tubule cells and is inhibited by previous damage to them. Probably half the dye taken up by the tubule cells at the beginning is secreted into the tubular lumen in the course of the next twenty-four hours; the remainder disappears, probably as a result of intracellular destruction. When a nephrotoxic dose of dye is given, the process of absorption and excretion appears to be the same, but the cells are damaged by the large amount of dye which they accumulate in their cytoplasm. The excretion in the urine is decreased in proportion to the extent of the lesions, and there is thus a prolonged retention of this large amount of dye in the cells, which probably continues and completes the initial damage.

Sarcoma of the Peritoneum.

T. F. HAVER (*The Journal of Pathology and Bacteriology*, September, 1932) reports a sarcoma of the peritoneum occurring in a man, aged forty years, who gave a history of abdominal pain and constipation, preceded for a few hours by faintness and malaise. On the sixth day signs of paraplegia were noted. A diagnosis of compression paraplegia caused by a tumour in the lower dorsal region was made. During the ten days preceding death the patient had occasional convulsions and passed into a uræmic state. *Post mortem* examination revealed extensive infiltration of the peritoneum, ureters and renal pelves with a pale grey tumour. An extradural, intraspinal metastasis was responsible for the paraplegia. The tumour was a pure round-celled sarcoma. The author states that this condition represents a true tumour of the connective tissue cells of the peritoneum and is not related in any way to the various types of retroperitoneal tumour.

Defence Reactions in Leuchæmia.

R. H. JAFFÉ (*Archives of Pathology*, August, 1932) has studied the inflammatory defence reactions in leuchæmia. He bases his discussion on the findings in ten cases of leuchæmia in which inflammatory lesions were found at autopsy and in which autopsy was performed so shortly after death as to secure perfect fixation. He finds that the type of response depends on the presence of myeloid tissue capable of producing mature granulocytes. Provided some myeloid tissue capable of maturation is left, the leuchæmic organism reacts to an infection like a normal one. Even in a diffusely lymphatic marrow the myeloid cells can proliferate and encroach on the lymphatic tissue when stimulated by an infection. If the granulopoiesis is completely exhausted, no defence reaction to an infection occurs. The author could not confirm the statements of other authors that leuchæmic cells may take the place of mature granulocytes; he found that very few immature cells appeared in the inflamed areas. It is generally accepted that the granulated cells of an exudate come from the blood. Extramedullary granulopoiesis, however, does occur under certain abnormal conditions, and, though in some instances it may result from the colonization of immature bone marrow cells, in the majority of cases the cells are formed locally. In the normal organism the histiocyte and the fibrocyte are unable to change into hæmocytoblasts. In leuchæmia, however, the range of cells that may develop into blood cells is much wider. The author has found in previous investigations that histiocytes may be transformed into myeloid cells in leuchæmia; from his observations of inflamed tissue he is convinced that the fibrocyte may also do so. He has been unable to find evidence for a reversion of the leuchæmic cells into fibrocytes or for their transformation into other cells (macrophages). He points out that the literature contains many reports of such transformations obtained *in vitro*. From this discrepancy between the findings inside the body and in tissue culture he emphasizes the need for care in drawing conclusions from observations made on explanted cells. The author makes passing reference to the conception that leuchæmia may be regarded as belonging to the group of malignant tumours. This conception is difficult to reconcile with the return of normal blood findings in leuchæmia during or after an intercurrent infection.

Tumours of the Thymus.

H. HANDI AND HALIS (*Virchow's Archiv für Pathologische Anatomie und Physiologie und für Klinische Medizin*, April, 1932) report the occurrence of three mediastinal growths arising from the thymus. They occurred in three male patients, aged respectively forty-five, sixty-one and fifty-three years. The macroscopic appearances at *post mortem* examination are described. In one instance metastases were found in the liver and left kidney, and in another in the mesenteric lymphatic glands, pancreas and suprarenal bodies. The microscopic appearances of two of the tumours is given in considerable detail. The growths consisted of lobules similar to those of the thymus. The lobules consisted of ridges of large cells and between the ridges were small collections of round cells. The authors discuss the origin of these round cells and conclude that small celled growths of the thymus have an epithelial origin, an epithelial structure and epithelial characteristics, in spite of the fact that on the whole their cells have the appearance of mesenchymal cells. These tumours therefore have a twofold nature. The authors state that they are justified in calling them thymocytomata.

Acute Pyæmic Cholecystitis.

K. TERPLAN AND S. SANES (*Archives of Pathology*, August, 1932) report a case of acute pyæmic cholecystitis. The patient was a woman, aged fifty-

eight years, who was admitted to hospital suffering from what appeared to be acute cholecystitis. *Diabetes mellitus* of moderate severity was found to be present, and blood culture yielded six colonies of *Staphylococcus aureus* per cubic centimetre. The primary lesion was acute osteomyelitis of the lumbar vertebræ. The internal surface of the gall-bladder was the site of superficial erosions and ulcers; acute pericholecystitis and a right subdiaphragmatic abscess were present. Histological examination of the wall of the gall-bladder revealed acute infarctions from bacterial emboli. The authors draw attention to the rarity of hæmatogenous infection of the gall-bladder in pyæmia and point out that in this case the symptoms referable to the pathological changes in the gall-bladder completely dominated the clinical picture.

MORPHOLOGY.

Ciliary Movement in the Nasal Cavity

A. M. LUCAS (*American Journal of Anatomy*, March 15, 1932) has made a detailed study of the nasal fossa as found in the monkey. Records of direction of ciliary movement were obtained from twenty-nine animals, of which seven were used also in measurements of rate of movement of particles. It was found that the nasal cavity of the monkey used (*Macacus rhesus*) is closely similar to that of man, except in certain minor differences which involve the superior and middle conchæ and the floor. In the monkey the maxillary is the only sinus present. The organization of ciliary activity in the nasal cavity is adapted to the complex structure of the nose in that the cilia propel mucus from the margin of olfactory epithelium. Ultimately all material carried by the ciliated respiratory epithelium of the nose is transported into the naso-pharynx. The degree of ciliary activity increases along a vertical axis from the roof to the floor. The average rate of movement on the medial face of the middle concha is given as 32·4μ per second, in the middle meatus as 50·7μ per second, in the inferior concha 52·7μ per second, and in the floor 110·7μ per second. As the mass of accumulated material becomes increasingly greater towards the floor, the regions in which particles are moved most rapidly are the same as those in which the mass of material is greatest.

The Development of the Hypophysis of the Cat.

S. BRAHMS (*American Journal of Anatomy*, May 15, 1932) states that the hypophysis is a structure determined by the cephalization of the vertebrate embryo. As such its origin is fundamentally the same at all levels of the vertebrate scale. Its development furnishes no evidence that it once was an exocrine gland. The hypophysis is an organ of purely ectodermal origin. Also, the hypophyseal angle or Rathke's pouch is not an active evagination, but is an expression of the total growth processes of the region attendant upon cephalization. Continued rotation and growth of the head material result in closure of the pouch to form the hypophyseal sac and the hypophyseal stalk. In the cat the ventral wall of the hypophyseal sac gives rise to the main body of the *pars distalis* (anterior lobe) by proliferation of its cells. The infundibular diverticulum arises as a depression in the floor of the diencephalon, at the apex of the hypophyseal angle. Evagination occurs and the ventral wall of the diverticulum comes to be related to the dorso-superior wall of the hypophyseal sac, that is, the true *Anlage* of the *pars neuralis* in contact with the true *Anlage* of the *pars intermedia*. The infundibular diverticulum comes to be surrounded by fused *pars intermedia* and *pars anterior*, with a potential lumen separating them. The *pars tuberalis* is at no time related to the *pars intermedia*.

The Hypothalamic Pupillo-Dilator Centre.

J. BEATTIE *et alii* (*Journal of Anatomy*, April, 1932) describe a series of experiments in which the central end of the severed sympathetic trunk was anastomosed to the peripheral end of a cut motor nerve in the neck. Recovery of muscular activity was noted in all animals allowed to survive for a period varying from 154 to 323 days. Stimulation of the pupillo-dilator centre in the hypothalamus produced the same type of muscular activity as stimulation of the cervical sympathetic trunk proximal to the anastomosis in animals surviving from 178 to 323 days. Evidence that motor impulses reach the paralysed muscles as early as the ninety-third day is recorded.

The Anatomy of the Intercostal Nerves.

F. DAVIES *et alii* (*Journal of Anatomy*, April, 1932) describe the muscles of the chest as four in number in each space: external intercostal, internal intercostal, intracostal and subcostal from without inwards. The subcostal muscle is variable in its extent, the others constant in each space. The intercostal nerves and vessels are confined to the plane between the internal intercostal and intracostal muscles. Each intercostal nerve divides at the back of the space into a series of branches: (*a*) the "main" nerve, which is prolonged as the anterior cutaneous branch, (*b*) the lateral cutaneous, (*c*) the collateral branch, which either rejoins the anterior cutaneous branch nerve or ends independently as a second anterior cutaneous branch. Communications between the intercostal nerve of one segment and the nerve above or below it in the series were also observed. The lower intercostal nerves form an elaborate plexus in the abdominal wall. Each muscle and skin "segment" receives branches from at least two, and more commonly three, spinal nerves.

Congenital Absence of the Kidney.

EDWARD BOYDEN (*Anatomical Record*, May 25, 1932) describes a young human embryo in which the kidney on one side is in process of disappearance and which thus provides, for the first time, direct information about the cause of this malformation in man. The defect seems to be due to an arrest in development of the caudal portion of the mesonephros, as a result of which the left Wolffian duct fails to reach the cloaca and therefore to give rise to the ureteric bud. In its absence the left blastema fused in this case with that of the right, to form a potential horseshoe kidney. More than three-fourths of the adult cases of renal agenesis are characterized by absence of the ureter. An account is also given of ureteral agenesis, experimentally produced in chick embryos by interrupting the Wolffian duct in its zone of differentiation. Despite the absence of the ureter, the blastema appears and assumes the form of an embryonic kidney, but fails to differentiate into tubules. An analysis of all data leads to the conclusion that no evidence has yet been presented which justifies the theory that absence of the kidney is due to lack of appearance of the metanephric blastema; on the contrary, it appears to be due to retardation in development of the ureter, to embryonic agenesis of the ureter, or to arrest in development of the parent tissue of the ureter.

True Congenital Solitary Kidney with Double Post-Renal Inferior Vena Cava.

A. MESSING AND M. F. ASHLEY-MONTAGU (*Anatomical Record*, July 25, 1932) give an account of a case of true congenital solitary kidney, that is, that condition in which the reno-ureteral apparatus has completely failed to develop on one side of the body, presenting thus only one kidney on the opposite side. The frequency of this condition is about one in 1,000, and apparently occurs with greater frequency in men than in women, and more frequently on the left than on the right side. In the case described the suprarenals are present on both sides and there is an associated anomaly displayed in the absence of the lower part of the inferior *vena cava*, as the right and left common iliac veins ascend to the level of the margin of the first lumbar vertebræ. Also on the side wanting the kidney there is an absence of the ureteral orifice, seminal vesicle, and ejaculatory duct. The scrotal portion of the *ductus deferens* was present and well formed, and this could not be traced beyond the abdominal inguinal ring. The testes on both sides were of normal size and appearance.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Royal North Shore Hospital of Sydney on June 9, 1932. The meeting took the form of a series of clinical demonstrations by the members of the honorary staff.

Cerebral Tumour or Status Epilepticus.

DR. F. GUY GRIFFITHS showed a female patient, aged fifty-two years, who was admitted to hospital on March 26, 1932. The patient had suffered from five attacks of acute rheumatism; the last attack occurred in 1929, and at that time the Wassermann test applied to the blood serum yielded no reaction. In 1921 the patient was subjected to the operation of salpingectomy, and in 1924 to thyreoidectomy. At the time of admission the right eye had been blind for many years. Right papillœdema was noted in 1926 and slight left papillœdema in the same year. For eight years the patient had occasionally had fits; recently these had occurred more frequently.

On admission the patient was comatose and the condition was thought to be due to uræmia. The patient was stout and had a puffy face. The right eye was glaucomatous. The systolic blood pressure was 154 and the diastolic pressure 124 millimetres of mercury. Slight albuminuria was present. The blood urea was 27 milligrammes *per centum*. The patient vomited a little. The knee jerks were absent and Babinski's sign was present.

After admission the stupor became less and the patient appeared, except for the glaucoma, to be almost normal. The condition of the eye improved with the use of hot foments and pilocarpine drops (eserine was used later). Lumbar puncture on April 5, 1932, yielded eight cubic centimetres of clear fluid under slightly increased pressure. The fluid contained less than three cells per cubic millimetre and its protein content was 150 milligrammes *per centum*. The hot packs did no more good and their application was stopped when uræmia was excluded from the diagnosis. One or two slight fits occurred, and three weeks later there were several more fits, which were epileptiform. During one of the fits, which lasted for one to two minutes, the patient was unconscious, the breathing was stertorous, cyanosis was present, the arms were held in flexion, convulsive twitches were present, there was no rigidity; the head and eyes were turned to the right; the pupil on the left side was semi-dilated and reacted to light; no facial paresis or spasm was noted; the plantar reflexes were extensor; a state of coma lasted for from seven to ten minutes and then the patient was stuporose. After a fit the patient was sometimes stupid and incoherent for a day or two. On one occasion she had an hallucination of blue beads and a blue dress, and then she regained her senses and her composure. The left pupil was regular and active; the cranial nerves were all intact, except for the double papillœdema. Sensation was unimpaired. The knee jerks and ankle jerks were absent and the plantar reflex was flexor. The patient had no paralysis, no rigidity, no flaccidity and no ataxia. She was treated with thyreoid extract in doses of 0·03 gramme (half a grain) three times a day without effect, and later on was given large doses of iodide of potash, up to 2·4 grammes (forty grains) three times a day, also without effect.

Throughout the patient's illness the temperature and respiration were normal, and also the pulse (except temporarily after lumbar puncture or other disturbance). The slight albuminuria continued, but there was no increase of blood urea or other sign of renal disease, and neither the blood nor the cerebro-spinal fluid gave a reaction to the Wassermann test. X ray examination of the skull revealed no abnormality. It was pointed out that Dr. Temple Smith and Dr MacDonald had attended to the ocular condition. The glaucoma subsided. Atrophy of the right disk was present and the eye was blind. There was papillœdema of the left eye, and the patient could do no better than read very large type with difficulty; sometimes her vision was restricted to the counting of fingers.

Pulmonary Tuberculosis and Tubo-Ovarian Abscess.

DR. H. Z. THROSBY showed a female patient, aged twenty-two years, who was admitted to hospital on May 10, 1932, complaining of pain in the right side and in the back of four years' duration. The patient's mother died of pulmonary tuberculosis when the patient was three months old, and one of her cousins had pulmonary tuberculosis. The patient had had one child four years ago and no miscarriages. The patient had suffered from chronic cough all her life. Her condition was diagnosed as pulmonary tuberculosis and she was sent to Waterfall Sanatorium four years ago. At the time of her admission to hospital she said that she had lost about half a stone in weight during the last two weeks; she was very breathless on exertion. Her general appearance was healthy. Examination of her lungs revealed tubular breath sounds and an impaired percussion note over both apices and over half of the left lung. The abdomen was soft and some tenderness was present in the right iliac fossa on deep palpation. On vaginal examination tenderness was discovered in both lateral fornices. Bacteriological examination revealed the presence of a Neisserian infection and tubercle bacilli were present in the sputum. X ray examination revealed chronic tuberculous changes in both lungs.

On May 12, 1932, under ethylene anæsthesia, subtotal hysterectomy with bilateral salpingectomy and oophorectomy was performed. Except for some attacks of breathlessness the patient had an uninterrupted convalescence.

Coarctation of the Aorta.

DR. W. W. INGRAM showed a male patient, aged twenty-five years, who had been a railway porter and a carrier for eight years without experiencing any discomfort. The patient had pneumonia when he was seven years of age and played games at school without any ill-effects. Two months ago he lifted a heavy weight and this was followed by breathlessness and by pain in the left shoulder, arm and back. Dr. Ingram pointed out that a diffuse pulsation was present in both clavicular areas. A large number of arteries were seen pulsating down the back. There was no pulsation in the lower limbs. The patient's systolic blood pressure was 230 and his diastolic pressure 180 millimetres of mercury. Calcification of the arteries was present. X ray examination revealed large, tortuous intercostal arteries. The patient's blood serum did not react to the Wassermann test. Dr. Ingram intimated that he hoped to report this case more fully at a later date.

Familial Muscular Dystrophy.

Dr. Ingram also showed a female patient, aged eighteen years, who was suffering from familial muscular dystrophy. The family history showed that the patient's mother and four maternal aunts had always had clawing of the hands; three sisters suffered from the same condition. The patient was admitted to hospital with clawing of the fourth and fifth fingers of both hands, with weakness of the whole of the left arm, with numbness of the hands and pain down the outer side of the upper part of the arm. She could not extend the interphalangeal joints of the fourth and fifth fingers. Wasting was present in both hypothenar muscles and in the interosseous muscles, especially of the left hand. At the time of the patient's admission to hospital there was no response to the faradic current from the interosseous muscles of the left hand, but at the time of the meeting a definite response occurred. There was general loss of sensation over the distribution of the ulnar nerve in the left hand, but sensation had been recovered. X ray examination revealed slight scoliosis of the spine in the cervical and upper dorsal regions, but no cervical rib. For twelve months prior to her admission to hospital the patient had been carrying heavy weights and her condition had improved very much with rest. Dr. Ingram said that he thought the condition was due to pressure by a fibrous band on the lower cord of the brachial plexus.

Cerebro-Spinal Syphilis Simulating Syringomyelia.

Dr. Ingram also showed a male patient, aged forty-six years, who had complained of a staggering gait for the previous six months. He had had several attacks of vertigo seven years earlier. In addition to his staggering

gait his symptoms included numbness and whiteness of the fingers of four months' duration. He wore out the toe of his left shoe. He had slight costiveness. He could thread a needle. He did not lose his balance and he could walk in the dark. He had no vesical disturbance. He had not burned himself unwittingly and he gave evidence of no mental change. He had spastic gait. The right and left tendon reflexes were exaggerated. He had an ankle and patellar clonus on both sides. The abdominal and cremaster reflexes were absent on both sides. The plantar reflexes were extensor in type. Wasting was present in the interossei and lumbrical muscles and in the thenar and hypothenar eminences. The fingers were white. The grip of both hands was weak. The dorsiflexors of the left wrist were particularly weak. The pupils were normal; there was no nystagmus. Speech was normal. There was no adiadochokinesis. Neither Rombergism nor ataxia was present. Sensation to sharp and blunt instruments was greatly impaired over the same area and in a small area immediately above and to the outer side of each elbow. Sensation to heat and cold was lost in the fingers. It was greatly impaired on the ulnar half of each forearm, over the shin bones and along the course of the cutaneous nerve from the seventh dorsal segment on the left side. The face was unaffected. There was no delayed sensation. There was no allocheiria. Both the blood and the cerebro-spinal fluid gave a "+++" reaction to the Wassermann test. The cerebro-spinal fluid contained twenty-two cells per cubic millimetre. It gave a positive reaction to the globulin test and the response to the bicoloured guaiac test was expressed by the following figures: 4444441000.

Acromegaly.

Dr. Ingram's last patient was a youth, aged eighteen years, who was suffering from acromegaly. The patient had suffered from rheumatoid arthritis for fifteen months. Six months before the meeting the patient began to need size eleven in shoes; he had worn size ten for several years. Puberty was established at the age of fourteen. The patient's temperament was normal, his appetite was good, and he had not wasted. His hands, feet, face and genitals were large. His cerebration was inclined to be slow. His basal metabolic rate was +29%. The sugar tolerance test yielded the following result:

(*a*) Before draught	0·09%
(*b*) Half an hour after draught	0·113%
(*c*) One hour after draught	0·20%
(*d*) One and a half hours after draught ..	0·17%
(*e*) Two hours after draught	0·13%

There was no glycosuria. X ray examination revealed an enlarged *sella turcica*.

Abnormality of the Pharyngeal Arches.

DR. BASIL RILEY showed a girl, aged three years, who was brought to hospital on account of asymmetry of the face, which had been present since birth. The left half of the face was smaller than the right. When the child was a fortnight old, a doctor had removed three "little" growths, one from the tragus and two from the cheek on the left side; two small pits remained on the cheek.

On examination the mandible was found to be the cause of the asymmetry, the vertical ramus being short and slender on the left side. The dentition, the mouth and nose and pharynx, the face, the ears, the head and the thyreoid gland appeared normal. In the myelo-hyoid region a body which was at first taken for an accessory thyreoid gland, was palpated.

X ray examination revealed the stunted left vertical ramus and also a half-moon shaped deficiency of bone, about one centimetre in diameter, in the posterior margin of the right vertical ramus. The body in the myelo-hyoid space appeared to be bony and like an accessory hyoid bone, larger than the proper one. There was nothing to suggest a syphilitic, hereditary or other ætiological factor.

The condition was demonstrated as an abnormal development of the mandibular arch and imperfect closure of the hyo-mandibular cleft, with resultant supernumerary pharyngeal arch between the mandibular and hyoid arches. The growths removed from the tragus and cheek were thought to have resulted from the displaced portions of the auricular *Anlage*. This was unfortunate in a girl, and Dr. Riley invited suggestions for treatment.

Foreign Body in the Brain.

DR. E. D. CLARK showed a boy, aged six years, who was struck by a motor car on March 24, 1932. The door handle of the car penetrated the frontal bone, entered the cranium and was snapped off its shaft. The child, on admission to hospital, was seen to have a small punctured wound of the forehead, just to the right of the mid-line, in which, almost level with the skin, could be seen a metal bar, apparently driven in horizontally and to the left at an angle of 30° to the median plane. He was conscious and answered questions intelligently, but was very irritable. The reflexes were brisk. Babinsky's sign was absent.

Dr. Clark, after preparing the skin, extracted the bar; this was not difficult. There was an immediate effusion of blood-stained fluid, then of blood. A strip of gauze was plugged into the cavity. A U-shaped skin flap was then dissected up; several loose chips of bone were removed; the gauze was pulled out a little way, then, hæmostasis being apparently satisfactory, the skin was sewn up with the gauze coming out through the wound.

On March 25 the gauze was removed. Signs of cerebral compression occurred, probably from œdema, and subsided in a few days. The wound became septic and was fomented. The brain now began to protrude more and more through the wound, which became very foul. Bilateral papillœdema was present, but there were no peripheral nervous signs.

On May 3 incisions were made into the scalp just behind the hernia, and irrigation through the wound was commenced. On May 15 a profuse discharge of thick pus from the left ear commenced and persisted. Dr. Clark pointed out that the brain hernia had shrunk rapidly and was a third of its former size; the child was the brightest and liveliest patient in the ward.

The door handle removed from the brain was 87 millimetres long, 15 millimetres wide, and 7 millimetres thick, and weighed 78 grammes (two and three-quarter ounces).

Diabetes Mellitus and Pulmonary Tuberculosis.

DR. COTTER HARVEY showed a male patient, aged sixty-three years, a hawker. This patient had been shown at a previous meeting of the Branch in 1931. He was then noted as having extensive pulmonary tuberculosis in the right lung and moderately severe *diabetes mellitus* (the fasting blood sugar level was 0·29%). Artificial pneumothorax was induced in April, 1931. The diet was fixed at 2,660 calories *per diem*, and thirty units of insulin were given per day. Dr. Harvey pointed out that twelve months ago the patient's right lung was well collapsed, his diabetes was under control, he was attending hospital as an out-patient, and his weight was 82 kilograms (thirteen stone two pounds). Since this time the patient had made steady progress. His pneumothorax had been maintained, but sputum had disappeared and the coughing was lessened. The patient was still afebrile and his erythrocyte sedimentation rate had remained in the vicinity of ten. No spread of the disease to the opposite lung had occurred. The patient had been kept on a line ration diet of 2,600 calories and was taking ten units of insulin twice a day. The urine had been sugar-free and the fasting blood sugar in the vicinity of 2%. For the past nine months the patient had followed his occupation as a hawker and walked many miles a day. During the year he had gained 18·9 kilograms (three stone) in weight.

Dr. Harvey said that the points of note in the case were the success of the combined treatment, artificial pneumothorax and high diet with insulin, and the possibility that insulin had contributed largely to the gain in weight.

Pulmonary Tuberculosis.

Dr. Harvey also showed a woman, aged thirty-five years, whose pulmonary tuberculosis had been treated by artificial pneumothorax and phrenic avulsion. The patient was shown at the Branch meeting at the Royal North

Shore Hospital in 1930. Artificial pneumothorax was first induced in September, 1927, for severe right-sided disease with cavitation. Fairly satisfactory collapse was obtained, though the apical cavity remained uncompressed. During the following two years the patient attended for refills and remained well. She gained 6·3 kilograms (one stone) in weight and now weighed 55·3 kilograms (eight stone eleven pounds). In July, 1929, the artificial pneumothorax was practically obliterated and further refills were not given. In October, 1929, the right phrenic nerve was avulsed by Dr. V. M. Coppleson, and since that time the diaphragm on the right side had remained paralysed. Soon after this radiological evidence of infiltration in the left lung was noted and the patient lost a little weight during the ensuing summer. Dr. Harvey said that when the patient was shown two years before, fear was expressed that the disease on the left side would spread and that hemiparalysis of the diaphragm would be a severe handicap for the patient. Subsequent progress had shown this fear to be groundless. The left side had improved considerably and the cavity in the right apex had shrunk. The patient's health had remained good, and during the two years she had gained 8·1 kilograms (eighteen pounds) in weight. Occasionally she coughed up sputum in which tubercle bacilli could be found. Dr. Harvey thought that the bacilli probably came from the small walled-off apical cavity. The points to which attention was drawn were the patient's successful progress and the shrinkage of an apical cavity as a result of phrenic avulsion.

Pulmonary Neoplasm Complicated by Pulmonary Abscess.

Dr. Harvey's next patient was a boiler-maker, aged thirty-three years, who, in Dr. Harvey's opinion, was probably suffering from pulmonary neoplasm complicated by pulmonary abscess. The patient first felt a little breathlessness in January, 1932. He soon developed a cough, at first dry and later productive of white, frothy sputum. For a time he complained of pain in the left infraclavicular region and could not lie on the left side. In April, 1932, the sputum became purulent and foul, and on several occasions the patient coughed up three or four ounces of blood. He had no fever, wasting, night sweats, or any other symptoms. There was no family history of chest trouble.

On examination there was, in the left side of the chest, above the heart, an area of impaired resonance, over which might be heard a few moist accompaniments to the breath sounds. Three X ray examinations had been made. On February 29 the radiologists reported the presence of an unresolved pneumonia in the left hilum. On April 15 the radiological diagnosis was probable tuberculous consolidation of the left infraclavicular region, with a small cavity and also great pleural thickening and enlargement of the hilar lymphatic glands. On April 29 the radiological appearances were suggestive of new growth. A lateral skiagram revealed a rounded mass in the neighbourhood of the bifurcation of the trachea and displacing the trachea forwards. No tubercle bacilli were found in the sputum, but fusiform bacilli and spirochætes were seen. The blood had failed to react to the Wassermann test. The patient was apparently in good health, except for cough, sputum and discomfort.

Thromboangiitis Obliterans.

DR. S. H. SCOUGALL showed a male patient, aged forty-one years, who was admitted to hospital on March 20, 1930, having complained of a painful left great toe for about one month. Swelling, aching and throbbing accompanied the pain. About one month prior to admission the toe-nail was removed in the out-patient department. This resulted in a temporary improvement, but the toe soon became more painful than before.

The patient denied venereal infection, but gave a history of moderate *potus*. He smoked a small quantity of tobacco. He had had no previous illnesses of importance.

The patient left hospital after four days with the toe still discharging and the condition was still present after eight weeks, in spite of local treatment. Some improvement occurred by October 15, when he was seen by Dr. Scougall. He complained of severe pain for some time, starting in the foot and going into the ankle, with colour changes to blue and white in the toes.

He was seen by Dr. Sinclair in consultation as possibly suffering from Raynaud's disease or arteriosclerosis. His systolic blood pressure was 158 and his diastolic pressure 100 millimetres of mercury. His heart and arteries were normal. There was no affection of the other foot or of either hand. Dr. Sinclair gave an opinion of arterial spasm in the vessels of the left leg and foot.

Teeth had been removed for pyorrhœa nine months previously. He had no sore throat. There was impaired sensation to hot and cold in the left foot, no ankle clonus was present, and the knee jerks were normal.

The patient then underwent a left lumbar sympathectomy on December 8, 1930, following which the toes and anterior part of the foot remained colder than the portion above, though he ceased to have colour changes. About one week after operation he complained of severe pain in the little toe, which was tender and painful to movement. Swelling increased, with blueness. At this time the toes were of good colour, and warm. He was discharged two weeks after the operation, with the pain diminished in the little toe and the colour improved (December 22, 1930).

In the following week the little toe became infected, necessitating readmission to hospital. The small toe was swollen and congested, with whitish patches. Pain was severe and the toe was quite warm, but not tender. The remainder of the foot was a good colour. The skin broke in several places, with a serous discharge. The severity of the pain was a feature.

On January 12, 1931, a periarterial sympathectomy was performed, but did not afford relief from pain, and conditions remained much as before. The patient was discharged on January 29.

As an out-patient he began to complain of cramp in the calf muscles after walking about a block. The pain never left the fifth toe, which later again became swollen and congested, and both feet became swollen. On March 27, 1931, an X ray examination revealed a loss of continuity in the middle phalanx of this toe, probably due to necrosis. The toe eventually healed about four months later. By this time there was absence of arterial pulsation in the *dorsalis pedis* and the posterior tibial artery of the left foot. Pain still remained in the fifth toe. On December 16 tobacco was stopped.

On January 15, 1932, the toe was still healed and there was hyperæmia of the foot, with tenderness and pain.

On May 13 pain was absent from the foot, but present in the calf on walking. There had been similar early signs and symptoms in the right foot.

Recurrent Dislocation of the Shoulder.

Dr. Scougall also showed a woman, aged forty years, whom he had seen on June 11, 1932. She gave a history of recurrent dislocation of the right shoulder since August, 1930. It occurred again in May following, and five times from then to September, 1931. The original dislocation was subcoracoid, due to a fall on to the right elbow when the elbow was posterior, giving the line of force as forward and upward. She had learned to replace it herself. The original dislocation had had immediate reduction and adequate fixation over the usual period. Once the condition was established, the patient was afraid to raise the arm to a right-angled abduction or to reach above her shoulder without fear of recurrence.

On September 2, 1931, a Claremont operation had been performed and recovery was uneventful, except for minor superficial sepsis. In the second week of December, 1931, there was a redislocation under the coracoid and it had occurred five times since operation. It was reducible by the patient with external rotation and forward flexion, and reduction was accomplished with a click. There was a little grating in the shoulder joint.

This patient was seen on January 11, 1932, and Nicola's operation was discussed, and two months later, after several more dislocations, this was performed on February 17, 1931. Dr. Scougall said it was interesting to find that the portion of the deltoid which had been brought round through the axilla, was definitely attached to the upper anterior portion of the deltoid.

Fixation was maintained for three weeks when mobilization was commenced. At six weeks full movements were allowed. Full function had been obtained and there had been no further tendency to dislocation.

Congenital Defect of Radius and Thumb.

Dr. Scougall showed a child who was suffering from a congenital defect of the radius and thumb. He said that the hand was fairly fixed in *manus vara* at birth, but within a month graduated correction by stretching had brought the hand around to normal alignment. For the present it might be possible to maintain this by occasional manipulation and a night splint. Ultimately several procedures were available: (i) the insertion of the ulna into a prepared socket in the centre of the carpus, with the wrist dorsally flexed; (ii) the use of a tibial bone graft obliquely across to the ulna to represent the lower end of the radius (Albee); (iii) the carpus might be inserted into the cleft lower end of the ulna.

Dr. Scougall quoted the following description by Jones and Lovett:

> This condition was more often single than double in 114 cases reported by Antonelli in 1905. The defect is generally not a total absence of bone, although superficially that may appear to be the case. Total absence was described by Kummel as present in the majority of cases, but the development of X-ray since that study makes the more recent data of Antonelli more reliable. When cases are carefully investigated, persistence of the proximal, with absence of the distal end is most often found, but absence of the middle portion may occur.
>
> There are often present anomalies or absence of the bones of the thumb and fingers, and perhaps muscular anomalies, and the radial artery may be absent. Club-hand (*manus vara*) is present in nearly all cases, with deformity to the radial side of the hand. Function may be fairly good or seriously impaired.

Practical Demonstrations.

DR. H. R. SEAR, DR. K. F. VICKERY and DR. B. P. ANDERSON STUART gave a demonstration of skiagrams. DR. E. C. TEMPLE SMITH and DR. W. M. C. MACDONALD demonstrated some ophthalmological operations. DR. R. J. SILVERTON and DR. C. M. EDWARDS performed suprapubic prostatectomy, demonstrating spinal analegsia with "Percaine" by a special technique, and a "new instrument" for facilitating hæmostatic suture of the prostatectomy cavity.

MEDICO-POLITICAL.

ANNUAL MEETING OF THE DELEGATES OF THE AFFILIATED LOCAL ASSOCIATIONS OF MEMBERS WITH THE COUNCIL OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION.

THE annual meeting of the delegates of the Local Associations affiliated with the New South Wales Branch of the British Medical Association with the Council of the Branch was held at the British Medical Association House, 137, Macquarie Street, Sydney, on October 7, 1932, DR. A. J. GIBSON, the President, in the chair.

The following delegates were present: Dr. R. W. Croft (Balmain District Medical Association), Dr. Kevin Byrne (Canterbury-Bankstown Medical Association), Dr. A. T. Roberts (Central Northern Medical Association), Dr. G. A. Buchanan (Central Southern Medical Association), Dr. K. S. M. Brown (Central Western Medical Association), Dr. A. M. Gledden (City Medical Association), Dr. W. T. J. Harris (Eastern Suburbs Medical Association), Dr. A. C. Thomas (Illawarra Suburbs Medical Association), Dr. H. R. Scrivener (Kuring-gai District Medical Association), Dr. R. J. Jackson (Northern District Medical Association), Dr. R. V. Graham (North-Eastern Medical Association), Dr. H. H. Lee (South-Eastern Medical Association), Dr. E. H. Loxton (South Sydney Medical Association), Dr. S. S. Rosebery (Warringah District Medical Association), Dr. J. T. Paton (Western Medical Association), Dr. T. Y. Nelson (Western Suburbs Medical Association).

Friendly Society Lodge Practice.

Proposals of the Friendly Societies' Association relating to the Common Form of Agreement were discussed. The proposals dealt with the following questions:

1. Extension of the metropolitan area.
2. City rates for City of Newcastle and suburbs, and municipalities outside the district of Sydney and suburbs and Newcastle and suburbs with a population of 7,500 and over.
3. Reduction of rates by 22½%.
4. That the Council of the Branch be asked to affirm the principle of agreements being made with central bodies instead of branches.
5. That the word "minimum" applying to rates be eliminated.
6. That fourteen days' grace be allowed for the supplying of medical lists.
7. That a common form of medical examination of candidates be adopted, such form to be drawn on the experience of insurance companies.

In regard to Clauses 11 and 26 of the Common Form of Agreement, Dr. E. H. Loxton moved and Dr. J. T. Paton seconded, *pro forma*:

> That the wives of friendly society members, when pregnant, applying for admission to medical benefits, be not admitted until thirty (30) days after their confinements.

It was resolved, by way of amendment, after a discussion, on the motion of Dr. E. H. Loxton, seconded by Dr. S. S. Rosebery:

> That the question of wives of friendly society members, when pregnant, applying for admission to medical benefits be referred to the Friendly Societies' Association.

In regard to Clause 26 (*a*) of the Common Form of Agreement, it was resolved, on the motion of Dr. K. Byrne, seconded by Dr. R. W. Croft:

> That in friendly societies contract practice, when a lodge secretary gives notice of a new member's name, he should also state whether the member's wife has been passed or refused admission to medical benefits.

Advertisement in Newspapers.

It was resolved, on the motion of Dr. R. V. Graham, seconded by Dr. R. J. Jackson:

> That the present By-Law 9 (*b*) is unnecessary and unduly restrictive and that it be replaced by the former By-Law 9 (*b*).

An Annual Meeting of Representatives.

It was moved by Dr. R. V. Graham and seconded by Dr. W. F. Simmons, on behalf of Dr. A. C. Thomas:

> That the Articles of Association and By-Laws of the New South Wales Branch, British Medical Association, be amended in order to confer on the Local Associations the status and powers of Divisions of the Association, and that the By-Laws and policy of the Branch be determined by an annual meeting of representatives of such Local Associations.

The motion was not carried.

Composition of Council.

It was moved by Dr. H. R. Scrivener and seconded by Dr. T. Y. Nelson:

> That this Association requests the Council of the British Medical Association to consider the possibility of reserving a certain number of seats on the Council for representatives of local and/or country associations.

The motion was not carried.

Eligibility for Membership.

It was moved by Dr. H. R. Scrivener and seconded by Dr. K. S. M. Brown, *pro forma*:

> That the By-Laws of the British Medical Association be amended to include the following:
>
> > That membership of the British Medical Association entail the proviso that members must be financial members of the Local Associations in the district in which they practise.

The motion lapsed.

Medical Practitioners Act.

A proposed amendment of the *Medical Practitioners Act*, 1915, was discussed. It was resolved, on the motion of Dr. W. T. J. Harris, seconded by Dr. S. S. Rosebery:

> That the time is now opportune for an amendment of the *Medical Practitioners Act* whereby unqualified persons should not be allowed to treat disease.

Baby Clinics.

It was moved by Dr. H. R. Scrivener and seconded by Dr. R. V. Graham, *pro forma*:

> That a deputation be arranged with the Minister of Health *re* the incorporation of the medical profession in the existing baby clinics as honorary medical officers in the districts where such baby clinics are situated.

The motion was not carried.

Workers' Compensation Act, 1926-29.

In connexion with the *Workers' Compensation Act*, 1926-29, the treatment of injured workers by medical officers of insurance companies was discussed. It was resolved, on the motion of Dr. A. T. Roberts, seconded by Dr. H. R. Scrivener:

> That medical officers of insurance companies and works should not treat patients sent to them without giving them an opportunity of being treated by their own doctors, if they so desire.

In connexion with By-Law 25 (i), i and ii, it was resolved, on the motion of Dr. H. R. Scrivener, seconded by Dr. K. S. M. Brown:

> That no member of the British Medical Association who is an insurance company medical officer be entitled to treat patients referred to the company by another medical practitioner in the course of accident insurance except the case be referred to the insurance medical officer through his own regular practice.

In connexion with massage and electro-physical treatment, it was moved by Dr. H. R. Scrivener and seconded by Dr. K. Byrne, *pro forma*:

> That members of the British Medical Association be advised to entrust their patients requiring massage and electro-physical treatment to qualified masseurs and electro-physicists in practice and not with the insurance companies.

The motion was not carried.

Schedule (Schedule "D") of fees and charges applicable where the medical attendant looks to the insurer for payment instead of exercising his legal right to look to his patient, or other person liable, in accordance with the arrangement of October 4, 1927, was discussed. It was resolved, on the motion of Dr. H. H. Lee, seconded by Dr. R. W. Croft:

> That this meeting requests the Council of the British Medical Association to endeavour to bring the Metropolitan Water, Sewerage and Drainage Board under Schedule "D" in regard to compensation cases.

Hospital Policy of the Branch.

The Chairman of the Hospitals Committee, Dr. R. B. Wade, addressed the meeting on the question of hospital policy.

Obituary.

PETER BRUCE BENNIE.

DR. PETER BRUCE BENNIE, who died at Sandringham, Victoria, on August 25, 1932, had lived in retirement for some years. The son of the late James Bennie, sometime civil servant and one of the pioneers of the Presbyterian Church in Victoria, Peter Bennie was born at Hamilton, Scotland, in 1852. He arrived in Melbourne with his parents when he was fourteen months old. His education was commenced at St. John's School (the old building is now part of the Presbyterian Ladies' College, East Melbourne) and became *dux*. He won an entrance scholarship to Scotch College in 1866, and in 1867, at the age of fifteen, became *dux* in classics, *dux* in English, and first *dux* of the college. He gained several exhibitions and went to the University of Melbourne to study arts. He graduated as Bachelor of Arts in 1871 and Master of Arts in 1872. He then accepted the position of Headmaster of South Yarra College to enable him to proceed with his medical course. He graduated as Bachelor of Medicine and Bachelor of Surgery in 1878 and took the degree of Doctor of Medicine in 1884. At school and university he was devoted to sport; he was an expert swimmer, won the high jump championship of the public schools, and for a time played football with the Melbourne Football Club.

After graduation in medicine he was appointed medical officer to the Daylesford Hospital in 1880 and began practice there. He came to North Melbourne in 1882 and began practice in Collins Street in 1883. During this year he joined the staff of the Children's Hospital and was associated with it for thirty-six years. At the time of his death he was honorary consulting surgeon to the hospital. He was an examiner at the University of Melbourne for many years in *materia medica* and therapeutics, and in dietetics and hygiene. He was President of the Victorian Branch of the British Medical Association in 1896 and President of the Melbourne Pædiatric Society in 1907. For some years he was Honorary Treasurer and Honorary Secretary of the Medical Defence Association of Victoria and Honorary Auditor of the Medical Benevolent Association of Victoria.

Bennie remained a student throughout his life. He published a book entitled "Rational and Effective Treatment of Hip Disease"; this was based on his experience gained during twenty-eight years of hospital practice. He published several papers in medical journals; among these were "Some Remarks on the Treatment of Diphtheria by Mercurial Salts" (1894) and "The Frequency and Intensity of Congenital Syphilitic Infection in Children" (1908). In addition to diseases of the hip he devoted special study to the operations for the cure of cleft palate and hare lip. Among his hobbies was the solving of chess problems and he won many prizes for solving problems in *The Leader* under the *nom de plume* of Petros.

Having lived for many years in retirement, Peter Bennie was not well known to the younger generation of practitioners, but he was one of those who by his clinical research has helped to mould surgical thought of the present day. His personal qualities are described by Dr. J. R. M. Thomson and Sir Colin MacKenzie.

Dr. J. R. M. Thomson writes:

My association with Peter Bruce Bennie goes back for many years. I remember, when I was a small boy at school, that he attained the position of *dux* of Scotch College and that his name was the first to be placed on the honour board which records the names of those who have won that position. He went to the Melbourne University soon afterwards, where he distinguished himself by doing a good course in arts, gaining many honours and coming out with the degree of Master of Arts. He paid particular attention to mathematics, as his thoughts leaned towards the exact sciences, and this he made of great service when he turned to the study of the medical sciences, as he did soon after the completion of his arts course. He applied the principles of the exact sciences in a practical way to medicine as few of us have done. As a

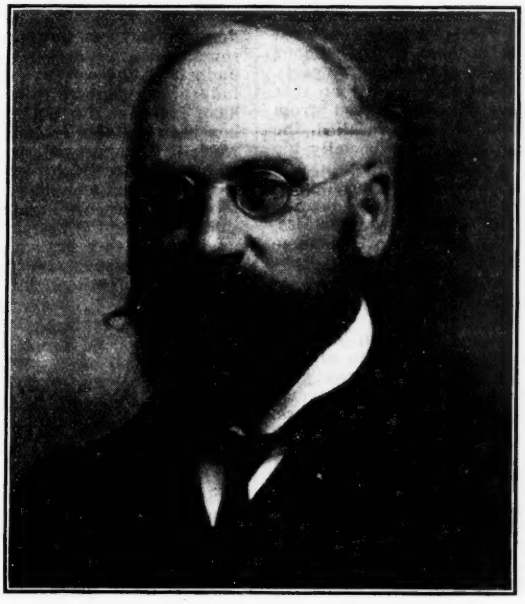
student he gained the esteem and affection of his fellows, so that he became that somewhat rare personage whom no one has a word to say against, but who yet is able to keep his estimate of himself within bounds by the exercise of a true humility. He had no touch of self-depreciation; he knew his own value and recognized it in a dignified way. He was generous almost to a fault. He never stood in the way of another's advancement, but rather effaced himself for the other's advantage. I don't know if it is necessary to make an apology for speaking in terms of eulogy of one who has so recently passed from us, but it is difficult to hold oneself back when thinking of Peter Bennie. His special scientific research work in the field of medical science has already been judged by the world, for his name has been placed among the foremost thinkers and workers in this branch of human knowledge.

Sir Colin MacKenzie writes:

With the passing of Dr. Peter Bennie there has been removed from our midst one of the great forces which have placed Australia in the front ranks of medical thought. His researches on the physiology and pathology of the living were mainly conducted among the patients at the Children's Hospital in Melbourne. To the present generation Bennie is best known in connexion with the conservative treatment of diseases of bones and joints, and especially that of hip disease. For this he slaved, he suffered, and he sacrificed. At a time when excisions and amputations were common, Bennie was able to write: "I have never seen a case in which amputation for hip disease was necessary, and if the joint is properly fixed, as by a Thomas splint, excision will never be required." This was not only a statement of fact, it was based on sound anatomical principles. Bennie knew that rest is the foe of tubercle, and that a joint, morphologically and physiologically, is dominated by the muscles which act upon it and which are its sentinels. In dealing with the question of immobilization for the purposes of conferring rest upon a diseased or damaged joint, the muscular factor should always be regarded as the important one. When we speak of "perfect" rest of a joint, whether to secure ankylosis on the one hand or to obtain improved or normal range of movement on the other, we refer to that position in which passive contact of damaged areas is reduced to a minimum by the proper disposition of the parts, and friction contact is eliminated by appropriate fixation. To rest a joint we must rest the muscles acting upon it. In the case of tubercle of the ankle joint, rest is not obtained unless the knee be rested also, and with a tuberculous knee the hip and ankle joints must also be immobilized. For the effective treatment of tuberculous disease of the hip Bennie made use of the Thomas splint. With him it was not a question of accommodating a deformed hip to the splint, but of the splint to the deformity. The splint, by means of simple wrenches which he devised, was accommodated to the zero position of rest for the diseased joint, which position the muscles will always strive to attain. In an advanced case this position might be one of acute flexion and adduction. With the subsidence of the disease the splint was gradually altered from adduction and flexion to what he considered the *desideratum* for function—absence of lordosis and of hip flexion. To implement these principles certain formulæ for the accurate fitting of the Thomas splint and the correction of deformities were used. The Thomas splint is the only one in the world whose application has been raised to a science. One formula, and the more important, is:

$$\text{Sin } \theta = \frac{\cos\alpha,\ \sin\beta}{\sqrt{(1 - \cos^2\alpha,\ \cos^2\beta)}}$$

where θ = angle through which the plane of the splint is rotated in the direction of the lateral displacement, α is angle of flexion required, calculated from 0° as normal, and β the angle of adduction or abduction, the rotation being made just above the joint and the flexion opposite the joint.



Bennie was a profound mathematician, and, like Sir Charles Bell, he looked on mathematics as the basis of all the sciences. To realize what this means to the teaching of anatomy one has only to compare the description of the cranium in a "modern" structural text book with that given from the point of view of function in Bell's "Anatomy", published over one hundred years ago. One would scarcely believe both were dealing with the same subject. According to Bennie's experience, tubercle treated by prolonged physiological rest showed little tendency to recurrence, in contrast to cases the subject of excision or amputation—and of this he had numerous instances. Bennie's text book dealing with the effective treatment of hip disease is worthy of a place in every practitioner's library.

Today little is heard of "growing pains", yet this was not the case when Bennie commenced practice. It remained for him to show, in a classical memoir published in America, that behind the use of that term were numerous conditions depending on disease and not on physiological growth, which were frequently overlooked, to the subsequent ruination of the victim.

On syphilis, as on tubercle, Bennie conducted numerous researches, and for years taught that tertiary lesions were infective, and that the disease was bacterial in origin. An examination, especially before operation, was never complete without an inspection of the big bones for nodes, the frontal bone for bosses, the presence of fissures, and the nature of healed wounds.

In these days of focal infections it is interesting to recall that over forty years ago an operation on the throat in a child was always preceded by an examination of the teeth and a visit to the dentist usually prescribed. Bennie had early recognized the importance of a septic focus in the body.

For the treatment of diphtheria today antitoxin is the basic remedy all over the world. Before its introduction Bennie wrote an important paper dealing with the use of frequently repeated and large doses of perchloride of mercury internally. Today mercury is still the most important of internal medications, and when introduced, compared with previous results, its effects could only be regarded as revolutionary. If science had not evolved an antitoxin, mercury would today be the basic treatment for diphtheria. Bennie advocated early intubation in preference to tracheotomy. Facts such as these the young practitioner, having antitoxin and the intubation tube at hand, is apt to forget. Bennie was a close personal friend of that distinguished Australian surgeon, the late Sir T. N. Fitzgerald, who was one of the world's great

exponents of subcutaneous surgery. If Listerism had not been introduced, the subcutaneous system would now be largely the vogue. Fitzgerald had an unbroken series of thirty successful cases of wiring for fractured patella, all of which had been treated by the subcutaneous method. His operation on *talipes varus*, which was a great advance in the treatment of this deformity, was also carried out subcutaneously.

Bennie was born and lived his life in the Commonwealth. At the age of sixteen he was the *dux* of the Scotch College, Melbourne, and he preceded his course in medicine by taking his Master of Arts degree. In addition to his knowledge of mathematics he was a distinguished linguist, and read the classics with ease. He studied anatomy and physiology under Professor Halford, the founder of the first medical school in the Southern Hemisphere. Halford was chosen for the Melbourne chair by Owen and Paget. Bennie obtained from Halford a first hand knowledge of the scientific upheaval associated with the publication of the "Origin of Species". This and the advent of Listerism had much to do with the introduction of the structural method of teaching anatomy to the detriment of function.

An incident of the Great War in 1917 is worthy of recall. Sir Robert Jones, nephew of the great Owen Thomas, had come down from Liverpool and established his orthopædic headquarters at Shepherd's Bush, in London. On the evening of the day on which His Majesty conferred a knighthood upon Sir Robert, a banquet in his honour was held at the Savoy Hotel. Members of the first party of American orthopædic surgeons entering the Great War were present, including Goldthwaite and Osgood from Boston. In his address Sir Robert Jones recalled the struggles the Liverpool School had had, and especially his uncle, in establishing conservative surgery in Britain, and he said that a big factor in his success was the support received from two great overseas surgeons, Peter Bennie, of Melbourne, and John Ridlon, of Chicago. I confess, as an old pupil of Bennie's, that the mention of his name under such surroundings gave me a feeling of no slight emotion.

Bennie can justly be regarded as the founder in Australia of conservative surgery in children.

As a pædiatrician he ranks with Trousseau, Henoch, Abraham Jacobi, and Thomson, all of whom he greatly admired. Like John Hunter, he was always ready to rearrange his ideas in accordance with scientific truth, even though such had been held sacred for many years. A few hours before his death he studied the latest number of the *Archives of Pediatrics* and of the *Archives de Médicine des Enfants*. Although aged eighty at his death, he left us when still young, cut off by misadventure. In the Australian Institute of Anatomy spaces have been reserved by the National Government for the purpose of honouring those great Australians who have advanced medical thought. Of Bennie's right to be so honoured, and to join such men as Bancroft, Thomas and Hamilton Russell, there can be no question.

HARRY LAWTON ASHBY.

WE regret to announce the death of Dr. Harry Lawton Ashby, which occurred on October 25, 1932, at Sydney, New South Wales.

Correspondence.

SQUINT AND SIGHT SAVING.

SIR: Being one of the few in Australia having any practical association and experience with the methods in an orthoptic clinic, may I suggest that a certain moderation be displayed in connexion with the establishment of clinics in the City of Sydney. It must not be thought that we have a new and ready cure for strabismus or for the cure of *amblyopia ex anopsia*.

Dr. Ringland Anderson (*vide* THE MEDICAL JOURNAL OF AUSTRALIA, October 15, 1932, page 495) confirms and emphasizes this point in the acute limitations of cases deemed desirable to treat and from whom some satisfactory results may be expected. Vision of at least $^{6}/_{12}$ could scarcely be regarded as an amblyopia within the ordinary and accepted interpretation of the term. The vast majority of squints and amblyopias are going to be excluded if such a limitation is to be placed upon admission to a course of treatment.

The modern instruments and apparatus are vast improvements over stereoscopes and amblyoscopes and the like, but are not wholly new and revolutionary. Nor is the method of their use a simple matter. On the contrary, it is a highly technical and painstaking business for which the kindergarten teacher temperament is essential. It would be a pity if every ophthalmologist felt that he must equip his rooms with muscle training and fusion centre training apparatus. The man with time to use them would scarcely have patients on whom to use them.

While I am enthusiastic about the whole idea and have to some extent pioneered the introduction of the apparatus, I believe it would be far more desirable to have a few hospital clinics, and good, rather than a mushroom growth at every hospital having an eye department.

Further, I would believe that the Ophthalmological Society of New South Wales would be well advised to assist in the establishment of a private clinic. Not as a financial interest, but rather as an advisory one, exercising some moral or actual control over the lady who might undertake the venture. Or perhaps by endeavouring to induce its members to support only such establishments as have satisfied the Society that the proprietor or director has been suitably trained and sufficiently experienced, will work to an approved scale of fees, will not advertise, and so on. I scarcely think we need as many lay practitioners as Melbourne now has in Collins Street, nor should we lose the present invaluable opportunity to hold some influence over the promotion of a private clinic. It behoves us to keep this branch of our work within some range of control or supervision and not let it be handled entirely by an unprofessional laity.

Yours, etc.,

GRANVILLE WADDY.

227, Macquarie Street,
Sydney,
October 22, 1932.

NURSES' REGISTRATION EXAMINATION.

SIR: In the October issue of the *Australasian Nurses' Journal* appear the five questions which constituted the paper on "Surgical Nursing" set in May, 1932, for the Registration Examination conducted by the Nurses' Registration Board of New South Wales.

As a large portion of the paper deals with subjects outside the realm of surgical nursing, one presumes that the above title was prefixed with the idea of preventing the candidates from falling into the error that they were answering a paper on "general medical and surgical knowledge" intended primarily for fourth year medical students.

On the other hand, perhaps the Nurses' Registration Board could explain the rationale by which such questions as:

a. Describe briefly the signs and symptoms of acute appendicitis.
b. Name the bones in the upper limb from shoulder to finger and thumb tips.
c. What are the signs (not symptoms) of fracture?
d. What is the line of the brachial artery?
e. What is meant by the following terms: (i) pernicious anæmia, (ii) complicated fracture, (iii) lordosis, (iv) spondylitis, (v) staphylorrhaphy?

fall within the realm of "surgical nursing"?

It would be just as logical to include in a paper on the above subject a few questions on entomology, palæontology, psychiatry, parasitology, dermatergoses, or even folklore.

One is forced to the conclusion that not only do the examiners fail to appreciate the limits and scope of

"surgical nursing", but they appear to be singularly unfitted to set a test paper for nurses on the above subject.

Yours, etc.,

H. RUTHERFORD DARLING.

229, Macquarie Street,
Sydney,
October 24, 1932.

AN ECONOMIC COMMENTARY AND DIAGNOSIS.

SIR: Doctors, being experts in diagnosis, should bring their powers to bear on the discovery of the causes underlying the present worldwide depression. It is quite certain that the politicians can do nothing effective until someone gives them a scientific diagnosis. The economists have failed in doing this, probably because they accept the present system and endeavour to improve its workings, rather than to alter the system itself. Most lay people, even the man in the street, recognize the necessity of a diagnosis. Medical analogies abound in their discussions of the problem, but no serious effort at making such a diagnosis seems to have been published, so that all political action has been of symptomatic or palliative character.

May I submit an attempt to deal with the problem on medical lines, in the hope that other doctors will help either to confirm or to confute the diagnosis?

Economics is the study of the mode of making, distributing and using the national livelihood. Agriculture is the typical economic operation, but sowing (labour) the seed (capital) in the soil (land) is not enough, it takes time to produce or destroy the crop.

Consumption, of course, is easy, dependent only on access to the goods, or on time alone. Production, however, involves labour, and at one time was apt to be insufficient, as evidenced by famines, shortages and so forth. Machinery has made production abundant. It seems clear, therefore, that the main fault in our system lies in the mode of distribution. Money is our chief means of distribution, our chief means of exchange. It is therefore to monetary matters that we need to give chief attention. But monetary reform cannot be successful without a comprehensive diagnosis of the underlying faults of our economic system. Money has a long history, going back to days when it was a definite thing—shells, cattle, beaverskins, tobacco, rum, gold, silver, precious stones. Since then it has become mainly a book entry, mainly overdrafts, with a token coinage for convenience in small transactions and a vault filled with gold for the anxious.

Agreement being reached on these points, we may proceed to the case for commentary in the hope of divining the root faults of our system.

We are now in the midst of a worldwide depression, and not for the first time: there was one in the nineties and one in the fifties of last century, to say nothing of minor depressions. Apart from recurrent depressions, our system is characterized by class feeling, strikes, lock-outs, rioting, fraud, profiteering, theft, and much other crime, besides sporadic unemployment, occasional bankruptcies, arrangements with creditors, constant loans, and universal fear of the economic future, coped with by pensions, charities and gaols.

In this depression we find universal indebtedness of primary industry coupled with unpayable returns and epidemic unemployment. We have restriction of output, destruction of what has been produced, while many of our families live on doles or sustenance, and underfed at that; there is the coexistence of poverty and glutted markets. Prices are very low and refuse to rise in spite of much doctoring of symptoms, such as high exchange, high or prohibitive tariffs, reduction of wages, cancellation of debts, lowering of interest, and increasing taxation. Rents fall, or fail altogether, dividends likewise. It pays better, really, to own war debts than to be venturesome in business, to grow or make anything, or to own property. Bankruptcies, reconstructions, moratoriums, arrangements with creditors are the order of the day. Repudiation is regarded as disgraceful, yet is practised under other names. The favourite way out seems to be to cancel old debts and then incur new ones.

During the war, when huge debts were created, there was much inflation. Since then there has been much deflation. Goods bought on a rising market have now to be paid for on a falling market. No wonder there has been conversion of loans on a big scale in more than one country. Our unit of money, the pound, is anything but stable, and has been as perilous to our economic system as a fluctuating inch would have been to the Sydney bridge.

The diagnosis is threefold:

1. The neglect of the effects of time: Our system is in discord with Nature. Nature each year gives us a crop which keeps us for the year, and gives us also the wherewithal to produce next year's crop. The nation as a whole lives each year on the year's crop; and that crop should be fairly distributed each year. This can be done if a money be devised which allows for the effects of time, which recognizes the wastage, as well as the increase by time. We need a four-dimensional economics—land, labour, capital and time. At present the wise man allows for depreciation, but the financier claims compound interest. We have no time limit to contracts, and permit one generation to bind its successors for ever. We know what time does to individuals—infancy, childhood, adolescence, adult age, middle age, senility—and to crops, and yet we ignore it in our national economy. No wonder that we get recurrent crises!

2. The use of debt as money: In Nature there is no debt; she gives us "sufficiency, surplus or shortage". The orthodox economist recognizes that all present day money is debt; but he does not seem to perceive that it is the use of debt as money that accounts for the universal indebtedness of primary industry, which in its turn is responsible for unemployment, for such indebtedness prevents the due exchange occurring between the primary and dependent industries. Until primary industry pays, unemployment cannot but persist; and primary industry cannot pay until debt is wiped out. Debt can only be wiped out by providing a credit money. Debt involves both repayment and interest. Debt therefore is really the private ownership of money; money belongs to the issuer instead of to the user. Our whole system of exchange between individuals and between nations is built up on money, and on money that belongs to others. That may have been reasonable when money was a real thing, but now that money is a mere book entry, it is both absurd and unnecessary. It is indeed the final stage of exploit through which the human race has passed. We have exchanged the warrior, the slave-raider, the feudal baron, the industrial "master" for the financier, at the same time enlarging the bounds of "wage slavery" until it includes all classes but the big financier.

Nearly all practical and theoretical men among the debtor nations realize the need of wiping out or reducing present debts and interest. But they seem to think that once debts are cancelled, new loans should be issued. So far, they have not seen that the debt system should be replaced by a better one—one employing a credit money, whose only obligation should be use.

3. The use of an elastic unit of money: hence inflation and deflation, with all their injustices. We not only use debt as money, but we permit the debt-owners to manipulate the value of the unit by which debts are measured.

Debt in the past has enslaved nations (ancient Egypt, Russia of 1597-1861), or has demoralized them by cancellations and doles (ancient Rome, present day England and Australia). Let us get rid of the debt system. [Readers should refer to Genesis, Ch. 41 and 47, and Leviticus, Ch. XXV, for the account of the enslavement of the Egyptians by debt, and of Moses's economic diagnosis and polity.]

Though this letter aims at the diagnosis rather than the treatment of the economic disease, it is perhaps permissible to mention that the Wice Currency, devised by Mr. L. G. De Garis, is based on a fixed unit of money (measured in labour time and valued in gold), allows for the effects of time, and is a credit money. It offers a very practicable treatment for the disease if once the diagnosis above given be accepted.

Yours, etc.,

MARY C. DE GARIS.

Geelong,
October 15, 1932.

Proceedings of the Australian Medical Boards.

VICTORIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1928, of Victoria, as duly qualified medical practitioners:

Flynn, John Michael, M.B., B.S., 1932 (Univ. Melbourne), 9, Martin Street, Elwood.

Ford, Edward, M.B., B.S., 1932 (Univ. Melbourne), 3, Moffatt Street, South Yarra, S.E.1.

Fortune, Cyril, M.B., B.S., 1932 (Univ. Melbourne), 7, Alfred Street, Coburg.

Graham, Keith Irving, M.B., B.S., 1932 (Univ. Melbourne), 410, Drummond Street, North Ballarat.

Green, Eileen Eva Hamill, M.B., B.S., 1932 (Univ. Melbourne), 49, Chrystobel Crescent, Glenferrie.

Griffiths, Leslie George, M.B., B.S., 1932 (Univ. Melbourne), 21, Adeney Avenue, East Kew, E.4.

Lowe, Thomas Edward, M.B., B.S., 1932 (Univ. Melbourne), 12, Haverbrack Avenue, Malvern.

Morris, Geoffrey Newman, M.B., B.S., 1932 (Univ. Melbourne), Trinity College, Carlton.

Murphy, Gregory Bernard Vincent, M.B., B.S., 1932 (Univ. Melbourne), 89, Studley Park Road, Kew.

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Niall, Desmond Paul, M.B., B.S., 1932 (Univ. Melbourne), 160, Church Street, Middle Brighton.

Penfold, Henry Boyd, M.B., B.S., 1932 (Univ. Melbourne), 53, Prospect Hill Road, Camberwell.

Provis, Ethel Lillian, M.B., B.S., 1932 (Univ. Melbourne), 13, Hemming Street, Moorabbin, S.20.

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Waite, Mary Elizabeth, M.B., B.S., 1924 (Univ. Melbourne), 169, Wattletree Road, Malvern.

Williams, Constance Isabel Arundel, M.B., B.S., 1932 (Univ. Melbourne), 59, Ulupna Road, Ormond, S.E.7.

Booth, Robert Philip, M.B., B.S., 1932 (Univ. Melbourne), Rosetta, Tasmania.

Additional diplomas registered:

Lawton, Frederick Donald Herbert Blois, F.R.C.P. (London), 1932.

Bearham, George Bankin, M.D., 1932 (Univ. Melbourne).

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xii.

LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officer (male).

ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, SYDNEY, NEW SOUTH WALES: Honorary Officers.

THE BRISBANE AND SOUTH COAST HOSPITALS BOARD, QUEENSLAND: Honorary Officers.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

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All communications should be addressed to "The Editor", THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.